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THE

PEDIGREE OF DISEASE,

BEING

SIX LECTURES

ON

TEMPERAMENT, IDIOSYNCRASY AND DIATHESIS

DELIVERED IN THE THEATRE OF THE ROYAL COLLEGE
OF SURGEONS IN THE SESSION OF 1881

BY

JONATHAN HUTCHINSON, F.R.S.

Late Professor of Surgery and Pathology in the College; Emeritus Professor of Surgery in the London Hospital; President of the Ophthalmological Society, etc., etc.

THE PEDIGREE OF DISEASE.

LECTURE I.

MR. PRESIDENT AND GENTLEMEN,—I fear I do not much mistake in the belief that the subjects which I have ventured to take for my present course of lectures are by no means high in professional favor. Our forefathers, who knew far less about the details of pathology than we do, attached far more importance to such matters as temperament and diathesis. They were accustomed to prescribe for a man's temperament; we think only of his disease, and turn aside with weariness from classifications of diathesis in which the physicians of an older day delighted. Although to a large extent this change of sentiment has been the result of advance in knowledge, yet I think it might easily be shown that it has gone too far, and that we now neglect unwisely the study of those differences between man and man of which, for the most part, physiology takes no cognizance, but which may yet prove of much importance in modifying the processes of disease. It is to this study that I now invite your attention. I have been attracted to it in part by its own intrinsic interest, and in part by the circumstance that it has seemed to be, in some sense, supplementary to the lectures which on former occasions I have had the honor to deliver from this chair. In my first course (three years ago) I investigated the present state of knowledge as to the influence of the nervous system in the production of disease. In the second we examined those remarkable and widely-spread forms of diathesis known respectively as Gout, Rheumatism, and Leprosy. When on these subjects I tried to show that rheumatism is a modification of the catarrhal diathesis, mainly nervous in its origin, in which the stress of the reflex disturbance falls upon the tissues of the joints. I traced a

close parallel between gout and leprosy, alleging that both are food-diatheses, being distinctly and definitely caused by certain peculiar articles of diet. Respecting both we had to remark upon the facts, that having been thus acquired by food, they became capable of transmission from parent to child, and that gout, at any rate, was prone to receive important modifications in such inheritance. In my third course, delivered last summer, I was led by a not unnatural sequence to take as my topic the laws of pathological inheritance in general, and to deal with them in relation to such maladies as syphilis, gout, leprosy, catarrh, and certain specialized defects, such as deaf-mutism, color-blindness, and hæmophilia. In each one of these courses I have been obliged very frequently to use the term "diathesis," and to justify its use against what I have all along recognized as a widespread and to some extent well-grounded distrust of the vagueness of the knowledge, or, shall I say, of the mere ignorance which its employment not unfrequently denotes. It has thus occurred to me that I could not take for the present course a subject more suitable than the one which I have announced. It will enable me to recapitulate, with amplification, some of the topics to which on former occasions I adverted briefly, to approach them from a different point of view, and, I trust, to place some of them in a clearer light. In doing this it will be my duty to avoid repetition, but if I should now and then seem to any of my audience who have honored me on former occasions to be attempting to enforce that concerning which I may be thought to have previously said enough, I must beg their indulgence. I must also offer some preliminary apology for the incompleteness and fragmentary character of my lectures. It will be impossible to pass even in the most cursory manner over the whole of the ground indicated, and I shall therefore prefer to dwell with some considerable detail upon certain portions of my subject with which I am personally familiar, and which also appear to have special value as illustrating general laws.

It is needful, in beginning, to define our terms, and in doing this I shall keep as closely as possible to customary usage. We may perhaps define the term *temperament* as applicable to the sum of the physical peculiarities of an individual, exclusive of all definite tendencies to disease. Different temperaments are to be assumed to be likely to give some degree of peculiar-

ity to morbid processes when such have been induced by other causes; but they do not in themselves involve any special proclivity. When most strongly marked, temperament is still consistent with the prolonged enjoyment of perfect health. If there be distinct proclivity, we must then use a stronger term, and speak of *diathesis;* and I would define a diathesis to be any bodily condition, however induced, in virtue of which the individual is, through a long period, or usually through the whole life, prone to suffer from some peculiar type of disease. Some diatheses are inherited, others are acquired. Of some, the effects are permanent or constant; of others, they are transitory, or recurrent after intervals of health. The term should, however, never be applied to any condition of health which is expected to pass away and leave no trace, for the idea of persistency, in some sense, is always implied. On the other hand, we do not confuse diathesis with dyscrasia, for while the latter definitely implies bad health, the former only denotes proclivity, and may be used when its subject seems perfectly well. Thus, to distinguish between temperament and diathesis, we may say that the former is a matter of physiology and the latter of disease, and that the former term is applicable only to peculiarities which are a part of the original organization of the individual, while the latter may be acquired as well as inherited. Thus, inherited diathesis is more often than not entirely latent at the time of birth, and is susceptible of aggravation, or, in some cases, of cure, in after-life. Such alternations are not possible in the constitutional peculiarities which we name as temperament.

Concerning the term *Idiosyncrasy,* I will not say more in the way of definition than that it is applicable to any definite peculiarity of organization of which the consequences may occur unexpectedly and otherwise inexplicably. It does not, like diathesis, imply any special proneness to disease, only that under certain well-known circumstances results which are peculiar to the individual will certainly occur. Although, as I shall have occasion to observe, it is quite possible that the idiosyncrasies may in many instances have sprung from the diatheses, yet they have become in long hereditary transmissions questions rather of organization than of disease. Their existence may, however, be revealed for the first time at any period of life, and nothing is more common than that an idio-

syncrasy should pass into absolute abeyance, to wait until, after it may be a very prolonged period, its proximate exciting cause may again make it conspicuous. Just as observers of mankind have from time to time strained their faculties in the endeavor to find in the external lineaments of a man some clue to his mental power and moral habits, so have physicians sought to find in parallel indications the means of predicting his tendencies in reference to disease. As yet I fear we must say that the labors of the physiognomist and those of the student of temperament have been alike disappointing. Whoever will set himself the task of attempting to classify a given number of individuals according to their temperaments will, I think, soon find himself baffled. He will discover that he is mistaking for criteria of temperament conditions which are simply indicative of youth or age, of health or disease, or the effects of past anxiety or trouble, or of comparative immunity from them. Just as the physiognomist ought carefully to exclude from his estimate of the original and inborn character of a man those traits of expression which have come to him through the wear and tear of life, and keep closely to the original features, so should the student of temperament scrupulously reject all that has been superadded, and which is in a sense accidental. Temperament, as I have endeavored to define it, concerns the original inherited organization of the individual, and does not include anything which is the result of the influences to which his life has exposed him. That which has accrued to him during life goes to produce or aggravate diathesis, but can do nothing in modification of temperament. Dr. Laycock has well spoken of the temperaments as being "fundamental modes of vital activity peculiar to individuals." To some such conception as this we must keep if we would use the word with any precision. Yet a very slight familiarity with the subject will easily show us how difficult it is so to restrict its use, and especially is this the case when dealing with adult and senile periods of life. The very names of the temperaments in many instances denote the record of the results of long-existing disease rather than of individual peculiarities as regards vital activity. Indeed, it may be questioned whether in a large majority of cases there do really exist in persons as yet in perfect health any peculiarities by which we can predicate or discriminate the "fundamental mode of vital activity."

Disease, when it occurs, will reveal it to us, and upon this point I shall have much to say in subsequent lectures. But I fear that so long as perfect health exists the data are exceedingly few and untrustworthy. When, for instance, we recognize a melancholic temperament, do we not usually take note of something which is the result of disease which has been experienced in the past, rather than of a peculiarity possessed by the individual while in health? The features which go to make up the sanguine temperament, are they not in part, and in large part, often those indicative of sound health in the digestive functions and blood-making organs, and such as might possibly be destroyed by a few years' illness? If we seek for something more fundamental, shall we not be obliged to confess that we have but little to guide us in a classification excepting the conditions which go to make up what we mean by complexion? In complexion we include the color of the hair and eyes, the state of the skin as regards thickness, thinness, or transparency, and the various degrees of freedom of distribution of blood in the capillaries of the face. It is easy to apply with tolerable accuracy such words as blonde, fair, dark, brunette, sallow, pale, florid, clear, muddy, and the like, and these and many others are epithets applicable to the complexion. Temperament, however, although to a large extent confessedly indicated by complexion, is generally held to include something more. If it did not I fear we should find it but a sorry basis upon which to build a knowledge of the vital peculiarities of the individual. Yet again, I ask, what have we to which we can make appeal? We may examine a man's features, note the size of his bones, the shape of his jaws, the brilliancy of his eye, the coarseness or fineness of his hair, his stature, his muscularity, his abundance or otherwise of cellular tissue and fat; but in observing all these things we shall be reminded that some of them are simply peculiarities of family, or of race, and have little or nothing to do with health, while others are conditions which may vary much at different periods during the same life. Above all, if I mistake not, we shall find it quite impossible to combine with well-known and indelible types of complexion any correlative peculiarities as regards the points which I have just enumerated. We shall find fair persons who have coarse skins, and others of the same tint of hair and eye who have transparent skins. We shall

find dark people who are florid, and others who are pale, and we shall have to note that the tendency to fatten varies very much at different periods of life, and that it often depends quite as much upon diet as upon temperament. I come then back to my point, and assert that whoever will set himself to classify by temperament a dozen healthy individuals whom he may chance to meet on a steamboat, in a law court, or at a dinner party, will find that he has scarcely any data excepting those of complexion. He will find, moreover, unless I am much mistaken, that if he attempts to go beyond mere complexion, there are not more than two or three in the dozen whom he can with any degree of confidence assign to special temperaments. As to complexion itself, the further he goes the more he will have to confess that, putting various conditions of sanguification aside as being in many persons dependent upon varying states of health, he can after all classify the complexions themselves only in reference to pigmentation. The varying degrees of dark, fair, or red constitute almost the sole individual peculiarities of the complexion which are not altered by disease or diet.

We come then to the question—Are congenital peculiarities as regards pigmentation in any degree trustworthy as indicative of vital peculiarities in the individual possessing them? We will ask the question first as regards individual races, and next as regards different individuals of the same race. That pigmentation as we now see it in the various races of man is an inherited quality and so definitely transmissible that it constitutes one of the bases of race distinction, may easily be admitted, while at the same time we must also grant that it is in itself a result of climate. Those who live in the tropics become dark, without question, as the result of the direct influence of the sun; while those who live in more temperate climates lose their pigment, or retain it only in certain special structures, as the hair, the irides, and the choroid. If apparent exceptions to this general law occur, they are to be met with probably as the consequences of human interference in modifying the conditions. Thus, the habit of wearing clothes prevents the development of any approach to blackness of skin in white races who may have lived through many generations in the tropics. The blackest skins always belong to those who have joined the two conditions of nakedness and a tropical sun. Thus, then, to begin at the beginning, may

we not believe that varying states of pigmentation of the integument denote rather exposure to climatic influences than peculiarities of development? Nor is there, so far as I know, much reason for believing that variations in this respect imply any material differences in health tendencies. Such peculiarities as we recognize in different races—the immunity of the negro from yellow fever, his liability to elephantiasis and to tetanus—may easily have nothing whatever to do with his pigmentation *per se*, and be matters simply of race. This view, I think, derives considerable support from the facts which I shall mention directly as regards the peculiarities of dark or fair complexions in members of the same race and among the lower animals.

If I have seemed to insist too much upon mere differences in complexion as being those to which almost all observers unconsciously trust in their endeavors to discriminate the temperaments in persons in good health, let me ask those who doubt it, to imagine themselves set to classify a group of negroes. It may be that the sameness in negroes is not nearly so great as it may appear to us, who are accustomed to deal with persons of very various types of complexion, and that those who have lived among them might be able to effect some sort of classification. We know that in a flock of sheep, which, to the untrained observer, all seem exactly alike, the shepherd can usually find distinctive individual differences. Admitting this, I still doubt, however, whether it would be practicable to apply to a race, in which all the individuals are highly pigmented, many of the facts which have hitherto been held to discriminate temperaments. We can recognize easily individual peculiarities — we can recognize also the peculiarities of race—but if the race be pure and the color uniform, we should have remarkably little to guide us. This statement leads obviously to the suggestion that I am perhaps involving myself in a fallacy, and that it may be the fact that what have been called the temperaments are observed almost solely under conditions in which the race is much mixed. It is certainly true that doctrines on this subject have been developed only under conditions of long-established civilization, and where the population represented those who derived their blood from various sources. Dr. Laycock has given a valuable hint in this direction, and has remarked that

we greatly need a British ethnology founded on well-defined and distinct characteristics. Thus, it might be, perhaps, much nearer the truth if we were to classify those who make up our own population not according to complexion, as fair, red, black, etc., nor by reference to certain more or less hypothetical vital tendencies, as lymphatic, bilious, sanguine, phlegmatic, and the like, but rather as Celtic, Scandinavian, Roman, or English. It is quite true that we should find a comparatively small number who could be definitely placed under any one of these heads, and that the large majority present features of racial mixture which would utterly defy classification. But the same difficulty has to be met by those who prefer to make groups under the head of temperament. A large majority belong to no one in particular, and partake of the characteristics of several at once. Modern historical authorities are desirous, I am aware, to have it believed that the English population is not mixed to anything like the extent which it was formerly the custom to suppose. They delight to claim us as *English* pure and simple, and to mean by the term direct, pure-blooded descendants of the Angles and the Saxons. They appeal to the fact of what is called the Saxon invasion—I must ask pardon, the *English* invasion—as to the entire extermination of the Romanized British population, from the land of which we are now in possession. They appeal also, and I must admit with more cogency, to the almost absolute substitution of a German language for the British tongue. There are, however, I think, good reasons why the student of race and believers in its permanency should hesitate to accept fully this new creed. We have among us far too many who possess not only typical Roman noses, but all the peculiarities of character which usually accompany the Roman cast of face. It is more difficult to feel certain as to the persistency of the British or Celtic element, since we know much less well what its peculiarities were. Whatever the evidence may be as to extermination by the invaders of the males who opposed them, nothing is less probable than that the women were included in the massacre. An army of invading and colonizing males would be certain to preserve sufficiently numerous representatives of the conquered race to effect a very material modification of the future stock. While on this subject it is also, I think, fair to suggest that the present frequency of the Ro-

man face in England, may not improbably be due in part to prepotency in that remarkable race.

Although I would by no means propose classification by race as likely to be of any great value in reference to clinical research, yet I do seriously think that it would be more practicable, and come nearer to the truth, than the classification by temperament. A glance at what has been attempted in the latter direction will, I think, convince us that even those who manifested the greatest ingenuity, and possessed the soundest and most extensive knowledge concerning health and disease, have been obliged, when describing the temperaments, to avail themselves of many criteria which are really the phenomena of disease. Chief among those to whom I allude stands one to whom I have myself been greatly indebted—my first teacher of medicine, the late Professor Laycock. With a power of insight which amounted almost to genius, Dr. Laycock had applied his mind to the study of the physiognomical diagnosis of disease, and by long practice in observations in this direction, he was enabled sometimes to give opinions which astonished those who were accustomed to more plodding methods of investigation. In a series of lectures delivered in 1861, and published in the following year in the "Medical Times and Gazette," he embodied the results of his experience. He believed that his rules of diagnosis were to a large extent based upon peculiarities of temperament, and he naturally gave great attention to the correct classification of these so-called fundamental states. For myself, I cannot doubt that the knowledge which he found so useful was based not so much on the discrimination of temperaments as on the recognition of diathesis. I could illustrate this statement by innumerable quotations from the lectures to which I have referred, in which features distinctly the result of bygone disease are mentioned, as if they formed part of the original organization. Our time will not permit that I should enter into any detail upon this point; but as Dr. Laycock's is, so far as I know, the last and certainly by far the best of all attempts to classify temperaments, I think it may be convenient that we should devote a few minutes to its consideration. He endeavored, as he tells us, to avoid the use of new terms, and to apply the old ones with definite meaning, as significant of predominant modes of vital activity. He recognized six divi-

sions—first, persons nervously active from predominant innervation; secondly, those with predominant sanguification and activity of the vascular and muscular systems; thirdly, those in whom both innervation and muscular activity are predominant, and this existing with also predominant carbon deposit or excretion—*the fibrous or bilious temperament;* fourthly, when the muscular system is well developed, but neither sanguification nor innervation predominant, and there is a decided tendency to the deposit of fat—*the phlegmatic temperament;* fifthly, those who are defective as regards innervation, sanguification, and muscular and vascular activity —*the lymphatic temperament;* and, sixthly, those in whom, with defective innervation, sanguification, and vascular activity, there is a combined tendency to carbon deposit—*the melancholic temperament.* Here we have, with a little expansion, the original four temperaments—sanguine, nervous bilious, and lymphatic. Let us ask if they are real, and if the features by which they are to be recognized are such as are likely to be permanent in the individual, and unmodified by alterations in his state of health. In the first place, I would suggest that the melancholic temperament and the bilious temperament are, after all, only different degrees of the same thing, and that, as life advances, the one is very apt to pass into the other; and, further, that the distinguishing feature in both is one which concerns disease rather than temperament, and which might be more conveniently known as the hepatic *diathesis.* It is the proneness to disordered function on the part of the liver, its ready and frequent occurrence, which for the most part stamps peculiarity on both these so-called temperaments. Further, it is much to be doubted whether this facility of hepatic disturbance would be found in association exclusively, or even generally, with any particular cast of the features, or, recognizable peculiarities in the general frame. Certainly it is not, by any means, the exclusive possession of dark-complexioned persons, nor, perhaps, does it more abound among dark-complexioned nations—the Italians, to wit—than among others. It is a matter of race, of family, and of climate. It may be diminished or aggravated by various conditions of life, and it is usually recognizable only after certain definite phenomena of disease have been already experienced. As regards its being worth while to distinguish any condition

as the nervous temperament because innervation is predominant—or, in other words, the central cerebro-spinal system is largely developed—much doubt may, I think, reasonably be felt. A high development of the nervous system is again a peculiarity of race and of family rather than of anything with which we as medical observers have to do. The conditions in which the nervous system is subject to disease are rather those of instability than of simple preponderance. The phlegmatic temperament gets its peculiarity from the tendency to fatten; but, as I have already observed, this tendency is shown in different periods of life, and is much modified by habits of diet and social surroundings. So of the lymphatic temperament, in which all functions are sluggish, and a general failure of what we may call "tone" is the conspicuous peculiarity. I would submit that this condition is not unfrequently induced by disease occurring during the lifetime of the individual, and not by anything which can possibly be denoted by peculiarities in the general formation. Dr. Laycock's classification of the temperaments is also, in common with the older ones, open to the criticism that, inasmuch as it takes little or no account of the more conspicuous differences in complexion, it affords us but few facts by which to recognize them. When a man is florid and muscular, it is not difficult to say that he is of the sanguine temperament; but these conditions are, perhaps, indicative rather of good digestion and sound health than of intrinsic peculiarities. The appetite may fail, or the health give way, and some of the signs supposed to denote temperament may easily vanish. Would it not be more convenient to speak of a man as being simply, for the time being, florid and muscular, than by the use of such a term as "sanguine temperament" to imply permanency in qualities which may easily prove to be otherwise? If we cannot identify the sanguine temperament by other characters, probably it would. The peculiarities which denote family and race must be viewed with the utmost caution, as well by the physiognomist in reference to character as by the physician in respect to tendencies to disease. They are usually of no use, and they may easily mislead. It is only indirectly that they may become valuable—that is, when the special proclivities of the race or family in question are known beforehand. A thick upper lip, when it occurs in a mulatto, denotes descent only, and not scrofula.

The peculiarities of the Hebrew features reveal only the race, and are of use to the physician only so far as he may be acquainted with the facts as to the special tendencies to disease in that race.

I have been speaking thus far on the subject of temperament considered as the aggregate of a man's physical personality, and have felt obliged to return the verdict that its study in this form is but little useful for our purposes as surgeons. It will I think, however, be well to say a few additional words respecting some of the individual peculiarities which have been held to make up temperament.

We will begin with the subject of *complexion.* The color of the hair, skin, and eyes may be easily observed, and although the English population, in a majority of cases, is a very mixed one, yet abundant opportunities occur of taking note of very definite types. Observations are yet wanting which would justify us in believing that any special tendencies to disease are implied by differences in the pigmentation of the hair and skin. It is, however, a subject of much interest for further research, and thinking that it might possibly be useful to others, I have placed before you a brief list of the more definite complexions which I prepared for my own guidance many years ago when investigating the subject of scrofula. In endeavoring to determine whether any given disease occurs in special connection with any one complexion, we are met at once with the difficulty that we do not know what the relative proportions of the several complexions are in the English population. Nor would it be sufficiently accurate for the purposes of any local observer to know the proportions of fair, rufous, dark, etc., in the British population generally, since these probably vary very considerably in different districts, and even in different towns. I cannot but think that careful statistics upon this head—collected in small towns and villages where it might be practicable to count the whole, and made, too, by trained observers—would prove of considerable value in more than one direction. It is in external diseases chiefly, such as lupus, psoriasis, and the like, of which the diagnosis is easy, that we should find the best opportunities for testing prevalence in relation to complexion. At one time I had formed a strong opinion that lupus, to take a marked example, was met with much more frequently in those of dark

complexions, persons of dark-brown hair, brown eyes, and a bluish sclerotic through which the pigment of the choroid is somewhat seen. I have collected a good deal of statistical evidence on this point, but it remains useless so long as we are ignorant of the relative proportions of the complexions among those from whom the cases came. It is probably the fact that both professional and lay observations concur in supporting the creed, that there are differences—mental, moral, and physical—between the fair and the dark; yet it would be exceedingly difficult to say anything definite as to what these differences are. I was myself taught by a careful practical observer, the late Mr. Wormald, that all persons of the dark complexion would bear mercurial treatment well, and would require larger doses than those who are fair. I have been accustomed ever since to act on this belief, and I think it is certainly well founded, although there are many exceptions to it. I have even gone farther than my teacher, and come to believe that a great many persons of the dark complexion not only bear mercury well, but enjoy better health while taking it, and are not unfrequently much and permanently benefited by a long course. It is but fair that I should admit, however, that one of the most remarkable examples of tolerance of this drug which I have ever met with, occurred in a patient of very fair complexion. Further, I have acquired, possibly on insufficient data, a sort of practical belief to the effect that dark-complexioned persons do not bear direct tonics well, that the need of purgatives is greater in them, and that they are often not helped by sea-air. Beyond these items of uncertain creed I cannot go, and even granting that they are sound, it remains still an open question whether the mere difference in pigmentation is, *per se*, their explanation. It may be after all that this difference in complexion is, as we see it in English society, mainly useful as a clue to descent, to family, and remotely to race. Many facts concur to imply this. When an albino chances to occur in a dark family (as is usual), he does not, I believe, show any special liabilities to disease, or differ in any way from his brothers and sisters, excepting in the local inconvenience as regards eyes and skin. White horses, white cattle, white rabbits, and white poultry are usually not possessed of any other correlative peculiarities. There is a prejudice, and probably it is nothing better, that they are slightly

more delicate than colored animals. The breed of pigs in the South of England is usually black, while in the northern counties white-skinned pigs are universally preferred. Such a fact is conclusive in proof that mere difference in pigmentation of the skin makes no material difference as to health or feeding capabilities. In the different breeds of sheep we have frequent illustrations of the power of race and family in giving peculiarity as regards the tolerance of climate and proclivity to some diseases. But the occurrence of a pigmented animal in any given breed does not, as far as I am aware, entail any difference as regards health. The only observation with which I am acquainted which contravenes this statement is one quoted by Mr. Darwin, to the effect that black sheep can eat with impunity a certain species of *Hypericum*, which is poisonous to white ones. This observation is so peculiar and so isolated that it must be held to require further confirmation. Possibly, the strongest instance in illustration of the unimportance of difference in general pigmentation occurs in the case of the common ferret. These animals breed albinos very readily, and probably three-fourths of those at present in use in England have red eyes and white hair, the remaining quarter being of a dark-brown color. So far as I know, the one is just as hardy as the other, and they are fed and treated in every way alike. Thus, then, I think we may believe that in the English climate the abundance or comparative absence of pigment in the tissues does not, *per se*, make any appreciable difference in the vital endowments of the individual. I am far from wishing to imply in saying this that the study of the complexions, as regards pigment, is useless for clinical purposes. Its uses have, however, for the most part, yet to be discovered, and it is desirable to note, in passing, that although we name the complexions chiefly in reference to pigmentation, something more than simply abundance or deficiency is to be recognized in them. Thus, the rufous complexion is due not merely to deficiency, but to peculiarity in quality, and is probably usually the result of the mixture of dissimilars. It is very likely that a careful observation of a larger number of rufous persons would confirm the popular belief that they do display peculiarities, not only in temper and general character, but to a certain extent as regards proclivity to disease. Again, the occurrence of great differ-

ences in color between the eyes and the hair, the eyes being much lighter than the hair—as, for instance, black hair with blue eyes—is probably an indication of delicacy. This, at any rate, is the general belief.

Next in importance to the differences in pigmentation we have certain other conditions which are usually included in what is meant by complexion, such as the thickness or thinness of skin, its transparency, the development of its glandular system, and the state of its appendages. I dare not venture to say more respecting most of these than that they are well worthy of further observation. We have exceedingly little definite information regarding the clinical value of any of them. We know that thick, coarse skins are more liable to be attacked by acne than are those which are thinner; or, rather, perhaps I ought to say that acne assumes different forms in relation to the original endowments of the skin which it attacks. In those of coarse skins, comedones, papules, and pustules easily form, or we may even have a condition of tuberous hypertrophy, while in those of thin, transparent skins, if the causes of acne come into operation, an erythematous condition will be produced, and the term *rosacea* become applicable. We believe—I scarcely dare say that we know—that in children in whom the hairs are thick and coarse the ringworm fungus finds an uncongenial home. It might be possible to mention a few other detached observations as to certain peculiarities in the skin and its appendages, not themselves due to disease, which may guide us in estimating the future liabilities of the individual. As a matter of general observation it may be asserted that all marked deviations from the normal type should be held in some degree of suspicion. Thus, if the hair is markedly coarse, or unusually fine, if the skin be very thick or very transparent, we are perhaps justified in suspecting that there may be in other tissues similar departures from the average constitution, some of which may prove inconsistent with the preservation of perfect health. For the present, however, it is speculation only. Of accurate observations we have none. The teeth and the nails may be suitably mentioned together as structures in which we very frequently observe with great interest and profit the indelible consequences of past disease. These, however, belong to the subject of diathesis, and do not

concern us now. As regards peculiarities of these structures occurring quite independently of previous disease, our knowledge is much in the same state as on the subject to which I have just referred. Peculiarities in the form of the teeth are often matters of family descent, and teach us nothing as regards their possessor's liabilities. It is probable, however, that all peculiarities as regards color and texture of teeth are of value, in proportion as they depart from the normal standard, as indicative of defective tissue formation in general. Dr. Laycock, in the interesting lectures to which I have alluded, has examined at much length the various conditions of the ear as indicative of temperament and diathesis. He believed that the form of the ear was of great value as a clue to the original development of the brain, and that the state of its nutrition, blood supply, etc., might help us to estimate corresponding conditions within the cranium. His conclusions in the main are probably correct, but we may doubt whether he did not push matters too far in assigning a special state of ear to such conditions as general paralysis, and in considering in reference to this disease that the congenital adhesion of the lobule to the cheek had some degree of significance. His observations had led him greatly to prefer ears which had free lobules, and these, too, of considerable size, which had all their different parts well marked, and which were moderately fleshy, not thin and cartilaginous. As regards the matter of the lobule, it may be plausibly suggested that its adhesion or otherwise indicates only family descent, and implies nothing as to temperament, and the same to some extent is probably true as regards the state of the helix and other parts. Making allowance, however, for sources of fallacy in these directions, I think there can be no doubt that the different states of the ear, as well congenital as acquired, have been unduly neglected both by the physiognomist and the physician. The ear affords excellent facilities for observation as to the general state of nutrition and circulation, and the degree of perfection in its form is a tolerably safe guide as to the descent of the individual from a sound and well-bred stock. That it furnishes a clue to the state of circulation in the brain better than does the color of the cheeks and nose, or anything like as good as that afforded by the brightness of the eyes, may be doubted, and before we make inferences on this point, we

must remember that the ears, as well as the nose, are remarkably exposed to the influence of external temperature.

I have said so much in disparagement as well of the general as of the special signs which have been held to indicate temperament, that I fear it may be suspected that I almost doubt the reality of temperament in itself. If I have given that impression let me hasten at once to remove it. There can be no question whatever as to the reality of the difference between individuals, nor any doubt as to the importance of the recognition of those differences by the medical practitioner. By far the commonest error of the prescriber, and one which most interferes with his success, is the easy-going habit of regarding all persons as alike, and recognizing differences only in their diseases; or, to put it in other language, of ignoring the predisposing causes, and taking account only of immediate ones. The farmer who would succeed in his pursuits must not content himself with making sure that he has sown good seed, and according to the most approved methods. He must go further back to take knowledge of the nature of the soil with which he has to deal, of the crops which it has previously borne, and of the manures which have been used. It is much the same with us in the diagnosis and treatment of disease. In addition to the primary or exciting cause, which is of paramount importance, we have various others which may perhaps be conveniently classed together under the term *contributory*, since they contribute to control and modify final results. Among these, temperament—the original vital endowment of the individual—is unquestionably a real force, and one which we would most gladly recognize and estimate if we could. The scepticism which I have been expressing applies not to the reality of the thing, but to our ability to discriminate it. I shall have to speak in my next lecture on the subject of idiosyncrasy, and one of the facts which I think will come before us in the clearest possible light is, that we possess no means whatever of recognizing by external configuration the subjects of these remarkable deviations from the normal state. Who shall discover beforehand the subject of hay asthma or the man in whom the iodide of potassium will act like a poison? When I come to the subject of diathesis we shall be on wholly different and far firmer ground. Defining diathesis as a persisting morbid proclivity we shall be able to show that in a

great many instances, either by the aspect of the individual, or the history of the case, a clear and definite opinion can be arrived at. The differences which have been held to constitute temperament have had their remote origin in two sources of influence; first, the hereditary transmission of the peculiarities incident to race and family, which, for the most part, entail no morbid proclivity whatever; and, secondly, to some one or more of the various common causes of disease acting, perhaps, through many generations. Thus, I cannot but think that what has been called temperament divides itself natually into these two parts, *race* and *diathesis*. There is therefore but little advantage in retaining the word, more especially when we have regard to the inextricable complexity of the subject. As regards diathesis, the conditions are very different. We can study the results of different causes in detail and with much precision; we can express our knowledge in clear terms, and recognizing the fact that we frequently encounter several of these causes in activity together, we can investigate with interest the mixed forms of diathesis which result. We do not embarrass ourselves by admitting extraneous complications based on the peculiarities of race. Making due allowance for acclimatization, we assert that, within certain limits,—not wholly unimportant, but relatively very much so—the various causes of disease act in the same way on individuals of very different races. It is in this direction, if I am not mistaken, that the work of the future will be done. We shall see that the connection between special diseases and the external configuration of the body is less close than has been assumed, and that the best plan is to study carefully the scope of power of each kind of morbid influence under the varied circumstances which may attend its action.

LECTURE II.

MR. PRESIDENT AND GENTLEMEN,—We concern ourselves to-day with some of the most remarkable facts in respect to what is known as *Idiosyncrasy.* In the definitions which were attempted in the previous lecture it was fully admitted, nay insisted on, that this word is intended to denote our ignorance of causes, though in no degree to express disbelief in their existence. Probably this ignorance will be, in most instances, only temporary, and the results which we need assign to idiosyncrasy and leave thus unexplained will, one by one, be transferred to the domain of the several diatheses concerning the causes of which something, at any rate, if not all, is known. Idiosyncrasy is, indeed, to a large extent, nothing but diathesis brought to a point. It is peculiarity of constitution in some one particular feature developed to a height which at first sight seems inexplicable and possibly almost absurd. It is individuality run mad. In seeking to understand the real nature of that with which under this name we have to deal, we must keep in mind that it is by no means always the isolated phenomenon which at first sight it appears. For one man who rises to the height of peculiarity which deserves the name *miser,* there are a thousand in whom the quality of thriftiness is developed in various degrees beyond what is praiseworthy. The miser is only the thrifty man developed in great excess. So it is with the relations between diathesis and idiosyncrasy; for one person who cannot take the smallest dose of quinine there are thousands who betray unusual susceptibility to the drug, and many of them in high degrees. Here, I think, we gain an insight into the way in which idiosyncrasies possibly take their origin. They are diatheses, or parts of diatheses, developed, intensified, and specialized by hereditary transmission. That they depend upon structural peculiarities we cannot doubt, although we may be quite unable to demonstrate their physical cause. The man who is

poisoned by a drop of atropine solution applied to his conjunctiva is as certainly the subject of some peculiarity in his nervous organization as are those who cannot distinguish red from green, or those who are born without the sense of hearing.

It may here be remarked that the term idiosyncrasy is not, I think, ever applied to acquired peculiarities, but solely to those that are congenital. Some, perhaps, may be overcome; many, as age advances, undergo modification, but, as a rule, all are present at birth, and persist through the whole of life. It is probable that all, when once developed, are hereditarily transmissible, and I am obliged to believe strongly that if inquiry were made we should, in almost all instances, find that all strongly-marked idiosyncrasies had been present in some degree in former generations. I suggested in last year's lectures, and with, I think, much plausibility, that the hemorrhagic diathesis—so strongly hereditary when once produced, unknown in the lower animals, and frequently coincident in the individual with gout—has its origin in the peculiarities of vascular structure which are developed by gout, and which have become modified and specialized by transmission through many generations. So I would venture now to ask consideration for similar hypotheses in reference to the other forms of idiosyncrasy of most, or of all, of which every fragment of genealogy has been lost.

We have defined idiosyncrasy to be a peculiarity of the individual, usually a rare and exceptional one, which does not necessarily entail any degree of proclivity to disease, but which may become obvious under a variety of exciting causes. It is often much as if by the introduction of some test solution, a chemist should discover the presence of chloride of sodium, or some other salt, in water which he had had no reason previously to suspect. The various incidents of life in connection with diet, drugs, and the exposure to the ordinary cause of disease, apply these tests for us, and most unexpected revelations do they sometimes make. We find that an individual apparently in no respect different from those around him, is poisoned by the smallest quantity of some ordinary drug, or that he cannot digest some one article of diet which is daily food to his companions; or, again, that the virus of some specific fever either produces on him no apparent effect, or may be attended by symptoms of tenfold their usual violence.

Sometimes it is an extraordinary immunity which is revealed, and sometimes an almost incredible degree of susceptibility.

We can never predicate anything as regards idiosyncrasies of function until the trial has been made. There are, however, a certain class of individual peculiarities of the same nature which do not always remain concealed. I allude to those in which the structure of some external and visible organ is affected. The peculiarities which we have hitherto classed as idiosyncrasies concern function only. But a moment's consideration will make it evident that they must depend on structure, and that they have really exact parallels in certain congenital defects or peculiarities in external organization, which may or may not entail inconvenience to the individual. A coloboma of the iris is a structural idiosyncrasy, so also is the persistence of the nerve sheath in the retinal nerve fibres in the human eye, which the ophthalmoscope occasionally reveals. And each of these peculiarities may, indeed must, entail some degree of deviation from the normal condition of vision. It is true that some congenital defects of structure occur in such an irregular manner that we prefer rather to class them, at any rate for the present, as mere accidents of development, or freaks of nature, than as idiosyncrasies; such are, for instance, moles, nævi, and the like. In the idiosyncrasies, however marvellous some of them may be, there is usually to be noticed a certain reference to law and order. They do not occur absolutely by chance, and they often appear to be correlated with other less startling peculiarities, and they often happen to several members of the same family, or are distinctly hereditary. I have instanced coloboma of the iris as an example of congenital idiosyncrasy of structure, and by its side I might put hare-lip, clefts in the eyelid, absence of the levator palpebræ, and many similar defects.

These congenital peculiarities of structure which do not entail any tendency to advancing disease of the whole organization, but remain permanently local, and must, therefore, rank as idiosyncrasies rather than diatheses, are not always apparent at the time of birth. Thus, a child may be born with its skin, so far as can be observed, in a healthy condition, but in whom within a few months of birth a harshness will be noticed which will steadily develop itself into the typical condition of ichthyosis. If this happened to but a single child in

a family we might search about for some cause of disease to which the infant had been subjected. But, if it happens to child after child in the same family, we cease our quest in this direction, and begin to feel certain that it must depend upon some congenital peculiarity in the structure of the skin itself.

Permit me next to ask your attention in some little detail to two groups of facts recently brought under my notice which will illustrate my argument. Mr. Balmanno Squire was kind enough last autumn to bring for my inspection a series of cases of most unusual interest. Very briefly the facts were these: Three children from the country, two sisters and a brother, of apparently healthy parentage, and having several other brothers and sisters who ailed nothing, were themselves the subjects of a malady the precise counterpart of which I had never seen before. It was exactly alike, with some minor differences of degree, in all the three children. Had it been a congenital deformity or arrest of development, its occurrence in several members of the same family would have been nothing remarkable, and we should have been able to place it, without any fresh wonderment at the marvels of pathogenesis, by the side of ichthyosis, color-blindness, deaf-mutism, and other well-recognized family diseases. But the malady in question by no means fitted closely with these. It had not been congenital in any one instance, but had developed in each child some years after birth. It consisted in an inflammation of the skin of the face and upper extremities, which had covered them with crusts and scabs, and which on some parts of the face, the nose especially, had caused ulcerations exactly like lupus. In fact, any one seeing the nose only would have had no hesitation in naming the disease lupus. But on the arms it was different. Here there was no evidence of new growth and very little of ulceration, but simply a diffuse, more or less pustular inflammation. From the occurrence of the disease on those parts most exposed to the air it was to be assumed that such exposure was the exciting cause. As it had not materially varied with season, and as the feet had not suffered from chilblains, we may conjecture that the irritating influences were wind and sun rather than direct cold. Let it be noted that in each of these three cases the disease had persisted for several years, and, allow me to repeat, that in all three children it was essentially alike.

What a lesson have we here as to the possibilities of hereditary diathetic proclivity! It may easily be the fact that not in the whole population of the British Isles, at the present moment, another case could be found which should offer anything approaching at all closely the disease I have described; and yet it had been possible, in the cases of three children, for the influence of parental combination of tendencies to be so definite and so peculiar, that they all developed it in precisely the same form. I have already mentioned ichthyosis as affording us an example of a disease which is often met with unexpectedly in many members of the same family, and probably, after all, it affords us the nearest parallel which we can find to these cases. It, however, in greater or less degree, is tolerably common, and generally there is some history of its having happened in former generations. It, too, is very often congenital, or if not actually present at the time of birth, it develops so soon afterwards, that we can easily believe that at any rate the peculiarity of skin giving tendency to it was congenital, and was of the nature of an arrest in the perfection of development. In the cases which I have described, however, the mind less easily grasps such an hypothesis, the disease being distinctly of an inflammatory kind, not affecting the whole surface, and not beginning until a considerable period after birth. Ichthyosis, for the most part, appears to be independent of local exciting causes, though it is by no means wholly so; and we must remember that in Mr. Squire's cases, although local causes are clearly influential, they have been so trivial in amount that we are obliged to place the inherited proclivities of the tissue in by far the foremost position.

Inherited proclivity of tissue of a similar kind, that is giving tendency at a certain age, under very insignificant exciting causes, to definite and peculiar forms of inflammation is probably a thing which we very frequently encounter in practice, but it is not often that we can demonstrate its reality in such a striking manner as Mr. Squire's series of cases enables us to do. It is chiefly on this account, although in itself an extreme rarity, yet teaching us a wide lesson, that I have thought it worth your attention. When we see tubercular disease of the lungs manifest itself in one after the other of a number of brothers and sisters without obvious cause, and run its course in each unchecked by treatment, we probably witness

an illustration of the same pathological law. It is to be remembered further that, although it is only when several members of the same family show tendency under the same circumstances to disease of the same part that the doctrine of inherited proclivity of tissue is strongly enforced, there is yet every reason to believe that the same cause is operative in countless examples of disease occurring only in single individuals. During the winter session just past two series of remarkable examples of xanthelasma in children, and in several members of the same family at the same age, have been brought before the Pathological Society. In one of these two brothers and a sister, now adults, were shown by Dr. Stephen Mackenzie, all three the subjects of xanthelasma in a slight but definite form, and all with the history that it had been noticed at birth. Mr. James Startin showed a brother and sister, still young children, in whom similar conditions were present in much more conspicuous forms, and in whom they had been noticed within a few years of birth, but were believed not to be congenital. In all these cases the changes are symmetrical, and occur in the limbs, neck, and trunk, omitting that part most usually affected when the disease developes, *de novo*, in adults, namely, the eyelids. In none is the affection attended by any derangement of general health, or tendency to liver disorder, and it seems probable from Dr. Mackenzie's cases that there is no tendency to increase, the three patients, now adults, having been in their present condition from early childhood. Thus, the general features of the malady differ exceedingly from those of the more common form as we see it in adults, and approach those of some other forms of skin disease—psoriasis and ichthyosis, for example. Histologically, however, the local changes in those cases are those of xanthelasma, and as such we must class them. In Mr. Startin's case the resemblance to psoriasis, as regards symmetry and location of patches on the tips of the elbows, etc., was very marked.

If speculation respecting such rare conditions may be permitted, I think it should proceed somewhat on these lines. We know, respecting psoriasis, that it is very hereditary, persisting through many generations, but rarely showing itself in more than a single member of the same family, and not unfrequently undergoing apparent transmutation into other

forms of skin disease, as, for instance, nummular eczema, lichen ruber, pemphigus, and ichthyosis.

Of its connection with the latter malady—parents the subject of psoriasis having ichthyotic children—I adduced some evidence in my last year's course of lectures. Of ichthyosis itself we know that it usually occurs in several members of the same family, is hereditary, but often omits a generation, or fails to descend in the direct line. Xanthelasma we have as yet known only as a disease which occurs chiefly on the eyelids of adults who have suffered from liver disorder, and which hardly ever in adults develops on other parts of the skin, excepting when in connection with jaundice. It is in part a new growth, and in part a fatty degeneration, and its peculiarities as regards color, etc., clearly have a close connection with the presence of bile pigments in the blood. I do not think that there can be much dispute as to the possibility of inheritance of any tendency to tissue change which has existed for a long time in the individual who was its first subject, and in whom it had showed itself a considerable time before the birth of offspring. Nor is it in the least certain, perhaps not even probable, that such hereditary tendency would be manifested in the first generation. If we apply these facts to the explanation of the occurrence of xanthelasma as a family, and almost as a congenital disease, apparently of no diathetic significance, I think we should conjecture that it is probably derived from some progenitor who had acquired the ordinary hepatic form of adults. And further, I cannot help the suggestion that it is possible that this inheritance has met and combined with a degree of proclivity to that group of skin diseases of which psoriasis is the best type.

Let me recapitulate: If a child shows within a year or two of birth yellow tubercles on its elbows, and delicate yellow streaks and spots in the flexures of the hips and popliteal spaces, exactly such as we are familiar with in connection with severe jaundice in the adult, and with jaundice only, we at first seek for evidence of disease of the liver. But if we find similar conditions produced at about the same age in two or three members of the same family, and the same series of events happening in more than one family, we again, as in the case of ichthyosis, cease our search for the ordinary causes of the disease, and fall back upon the hypothesis of what I

have ventured to term structural idiosyncrasy. We feel sure that the skin of the parts affected must have been at birth in a state of peculiarity, although we failed to be able to prove it. It is, of course, possible that the congenital peculiarity was not in the skin alone. It may have been in the blood also; but if we observe, in the subsequent life of the patient, that the peculiar conditions remain restricted to the parts first affected, that they have no aggressive power, and that the general health of the individual does not suffer in the least, our first impression as to the idiosyncrasy being purely one of local structure becomes revived and strengthened.

I am yet very anxious to make clear the argument that congenital structural peculiarity may entail liability to local disease, and that such disease may be locally aggressive—at any rate, for a certain time, perhaps indefinitely—because I think it is one which may, probably, explain a large class of pathological facts, some of which are of a far less definite character than those to which we have just referred. Let us use ichthyosis and xanthelasma of young children as definite and palpable illustrations of what is possible, although admittedly rare, and guided by the light which we obtain from their investigation, let us examine the facts as to some other much less definite maladies. In the condition known as molluscum fibrosum, little tumors—possibly fibro-cellular, possibly glandular—develop in or under the skin. Usually there are very few of them, and examples of the malady, when only two, three, or half-a-dozen are present, are very common. In rare cases, however, the whole surface is affected, and the tumors may be counted by hundreds, or even thousands. The tendency to their production may be noticed for the first time at almost any period of life, but if they are to be numerous, it begins almost invariably in early childhood. Whether there be many, or whether there be few, the general health remains good, and there is no evidence whatever of any correlated tendency to general disease. Is it not very probable that the liability to molluscum fibrosum is inbred in the original development of the integument, that we are witnessing the results of an idiosyncrasy of structure? Let us see how the matter stands as regards common psoriasis. In this instance we have no new growth of tumors, but only a tendency on the part of special regions of the skin, possibly of almost the

whole of it, to take on a certain peculiar type of chronic inflammation. This tendency occurs in its best marked forms, and most frequently to young persons, although, let us note, it is never congenital, and never even infantile. It is never associated with anything that we can appreciate as ill-health, and the facts with which we are acquainted as regards the conditions which may influence it for the worse, are few and insignificant. Excepting so far as it may be supposed to act by interfering with the functions of the skin, it does not produce any derangement of the patient's health. It is hereditary, but not prone to prevail in many members of a single family. Is it not probable that a disease, of which these statements are true, is one not originally or wholly due either to the blood or to the nervous system, but in large, and chief part, to an idiosyncrasy in the structure of the skin? In proportion as the disease is late in its development—in other words, in proportion as we find it apparently waiting for the exciting influence to assist its birth—may we assume the probability that the idiosyncrasy is less strong. The explanation of the entire absence of psoriasis in infancy and early childhood may possibly be found in the suggestion that, when the structural idiosyncrasy is very strong, it manifests itself in a different form. Thus, I think it not improbable that ichthyosis of infants may be in this way the representative of the psoriasis tendency in its intensest form. I must not weary you in arguing the case in reference to other diseases of the skin, or of other parts, but simply state that whenever we find a strictly local malady which develops itself in all instances with little or no apparent cause is hereditary, is but little aggressive, and only in a sort of accidental way influences the health of its subject, we are entitled to suspect structural idiosyncrasy. Steatomata on the scalp, lipomata, adenomatous tumors in the breast, multiple uterine fibroids, milium, whether on the face or elsewhere, and a host of others, are probably examples of what I have tried to describe. From these it needs but little ingenuity to perceive that it is only a matter of degree if we should pass to the development of new growths in general, both malignant and innocent, and to many disorders which do not fit at all exactly with the description which I have given and concerning which we can say a good deal as to our knowledge of their exciting causes. In a mul-

titude of these, hereditary, but wholly latent, peculiarity of structure may not improbably take its share in the production of the special result. They are examples of what may be termed complicated or conditional idiosyncrasy.

I must hasten to other departments of our subject. As surgeons we have to encounter the results of functional idiosyncrasy chiefly in reference to drugs and the specific fevers. The department of diet I ought, perhaps, to leave to physicians, yet it is impossible that I should wholly do so. For the patients who consult us about other maladies will often describe to us symptoms which we should wholly misinterpret were we not familiar with what is possible in reference to diet idiosyncrasy. A few years ago it fell to my lot to become much interested in the subject of idiosyncrasy as regards eggs. Several patients consulted me within a short period, who, among other symptoms, described a liability to attacks of violent vomiting, or of a sense of sinking and abdominal distress which were to them inexplicable. In more than one of these the reason of my being consulted was that the attacks were attended by temporary defect in sight. In one instance the patient was an artist, who declared that frequently he was quite unable, for several hours at a time, to see to paint. His defect seemed to be a temporary suspension of the power of accommodation. It always affected both eyes, and was always attended by a sense of heat at the stomach and abdominal discomfort. I found that these attacks usually occurred within an hour or two of breakfast, and on entering into detail I became convinced that they were due to eating eggs. On telling my patient my conclusion he replied at once that he had always suspected it, yet being very fond of eggs, he had indulged in one once in a month or so. He was quite cured by abstinence. In several of the cases to which I refer, the symptoms produced were far more violent than in this one, and had been so severe on some occasions as to lead to the suspicion that poison had been given. In some cases susceptibility as regards eggs is observed in reference to all eggs, and in all conditions as regards freshness, and in respect also to the very smallest quantity, so that a little egg taken in a pudding, a sauce, or a soup, may produce the most definite disagreement. In some the susceptibility is invariable, but in others it may be much diminished, or entirely removed when

its subject is in unusually good health, or taking much exercise in the open air. In many instances the liability to egg-poisoning is manifested only in respect to the eggs of certain birds, or eggs under certain special conditions as regards cookery or keeping. Egg and fish, the two articles of diet respecting which most is to be said as regards peculiar susceptibilities, are, it is to be remembered, the two which are most easily capable of peculiar changes from over-keeping. A fish which is quite wholesome when absolutely fresh may become exceedingly otherwise if kept only for a few hours after death, a fact with which those who live in hot countries are abundantly familiar. The same is true, though to a less extent, in respect to eggs. Eggs are perhaps as much influenced by methods of cooking as by mere keeping, and I may remark in passing that the examples of the most violent egg-poisoning which I have known have occurred to individuals who, having manifested the idiosyncrasy to some extent at home, had incautiously partaken of articles containing eggs while travelling abroad. Among various other articles of diet respecting which peculiar susceptibilities are frequently shown, we have honey, tea, coffee, various fruits and vegetables, and various forms of alcoholic beverages. Cases of what deserve to be known as *tea-poisoning* are probably by no means infrequent, but the medical adviser who, having become cognizant of some of these, should straightway proceed to discourage the use of this beverage by all, would be making a very wide departure alike from the laws of clinical induction and common sense. We have to deal with idiosyncrasy, and must apply our knowledge under the limitations of its laws. It must be remembered, however, as I have already asserted, that idiosyncrasy by no means declines to enter into partnership with other causes of functional disturbance more easy to be understood. The varying state of the patient's health at different periods of life, and varying habits as regards diet at different times, may conceal or bring into unusual prominence an idiosyncrasy which is itself a permanent condition. In many patients susceptibility to the influence of tea is much increased by taking certain wines, notably champagne, and is diminished by malt liquors and the red wines. As a rough rule, all idiosyncrasies become aggravated when the tone of the system is low, and many of them pass into almost

absolute abeyance under conditions of unusual vigor and health.

Idiosyncrasy in respect to tobacco displays itself in two forms, and one of them is of unusual interest, since it exemplifies a class of idiosyncrasies which are very apt to be misunderstood. I refer to those in which the individual susceptibility is not displayed immediately on the incidence of the exciting influence, but waits, it may be, for a long time, and until possibly other conditional causes come to its assistance. A little thought will probably supply most of us with examples of this class of idiosyncrasies, and they are very important in practice. A man who has drunk tea all his life, and largely, may rather suddenly find that it is almost poisonous to him, and may then recollect that one or more of his relations have never from childhood been able to take it. It is the same with eggs, fish, and certain kinds of wine, and so also with various drugs, and in all these instances it is, I believe, quite possible that the display of the idiosyncratic susceptibility may be only temporary. In some instances we may discover in some change in the mode of life or habits of diet, or in advancing age and possibly some visceral degeneration, an explanation of the newly-discovered peculiarity, but in other cases nothing can be found.

Tobacco, I think, stands curiously alone among the articles in human use in that it almost invariably in the first instance acts as a powerful poison, and that tolerance can be acquired quickly and certainly by almost every one. There are, however, great differences in the degree of susceptibility, and in the ease with which habituation is obtained. Many never do obtain it, and although this in most cases may be merely the result of want of perseverance, there is a certain number in whom it would appear that the primary idiosyncrasy is insuperable. Many persons who have acquired tolerance of one kind of tobacco remain susceptible to others, and after having been habitual smokers for years, may be made very ill by inadvertently using an unaccustomed kind. Many smokers, I believe, recognize periods during which the pipe disagrees with them, and they are obliged for a time to abandon it, and very curious symptoms, such as absolute suspension of the sexual appetite, etc., are sometimes with confidence alleged as consequences of its excessive use. But the cases which are of most

interest in connection with our present subject are those in which without any excess and in persons who have been for long thoroughly habituated, tobacco suddenly begins to act as a poison without causing any distaste or any conspicuous derangement. These patients are often quite unaware of what it is which is hurting them. They lose appetite and become nervous, and among the most conspicuous symptoms is a very marked failure of sight. This failure differs from all other forms of amaurotic amblyopia. It always affects simultaneously and equally both eyes; it begins very insidiously, and having no other symptom than diminution in acuity of vision, is often scarcely recognized by its subject until at the end of six weeks or two months he finds that he can only read the largest type. When at its height the optic disc is usually red and a little hazy, and at a later stage it becomes decidedly pale. If the patient will for a while leave off smoking he will regain his sight almost to a certainty, but, however perfect may be his apparent recovery, the disc is almost always left pale. He may resume the habit after awhile without much risk of relapse if he keeps to strict moderation, and in some cases recovery takes place, although the abstinence has never been complete.

Such are some of the principal facts respecting Tobacco Amaurosis. I have felt justified in stating them in this positive manner because I published in a series of reports some years ago in the "Ophthalmic Journal" and the Royal Medical and Chirurgical Society's "Transactions" very full details respecting them. That the symptoms in question are really due to tobacco I cannot feel the slightest doubt. I have seen no cases quite parallel to them either in women or in non-smoking men. If in these two classes of patients similar symptoms should be presented, which is very rare, I believe that they always progress without interruption to almost total blindness. Tobacco amaurosis rarely happens to novices in smoking, and seldom except to those who are liberal in the indulgence. Half an ounce of shag per day has been the usual allowance in the cases which I have collected. In support of my interpretation of the facts I may briefly advert to the history of our knowledge of them. Long before the ophthalmoscope-diagnosis was made out, Dr. Mackenzie, of Glasgow, and several French observers, had recognized the malady, and

alludlng to the difficulty met with in persuading its subjects to abandon the habit, Dr. Mackenzie had used the expression, "There are many who would rather smoke than see." Mr. Wordsworth, at Moorfields, was, I believe, one of the first who studied the opthalmoscope-diagnosis, and he held that the conditions displayed by the disc were pathognomonic. It was to his teaching that I was chiefly indebted for my first interest in the subject.

I must hasten to the narration of certain facts which show that this disease is really a matter of idiosyncrasy, and that we ought, by recognizing this fact, to explain the circumstance that such a very small proportion of smokers ever suffer from it. This immunity of the greater number and the picking out of one here and one there has been a great stumbling-block to many in the way of accepting this doctrine. I cannot think that it ought to have been so if due allowance be made for the influence of idiosyncrasy. The facts to which I first alluded as supporting the belief that this influence is really at work are the following: Not very unfrequently the subject of tobacco amaurosis is one who was well aware of his susceptibility, who had experienced unusual difficulty in learning to smoke, and who had never been able to change with impunity from one kind of tobacco to another. Some years ago a young man came under my care who was a smoker, and who had smokers' amaurosis. Subsequently his mother, who did not smoke, came to me for an almost similar state of things, and I then learned that a nephew of hers, a smoker, had suffered just like his cousin, and had been under the care of my colleague, Mr. Hulke. I applied to Mr. Hulke for his diagnosis, and he kindly sent me a copy of his notes, showing that he had considered it a tobacco case. Here, then, we have three near relations presenting the same form of disease. In the woman, who did not smoke, the disease did not show itself until twenty years later in life than in the two young men who did, and in them, on abstinence, the disease was arrested, while in her, in whom no change of habit could be made, it increased. Can we doubt that we have here an instance of family proclivity?

I have yet to mention a stronger case in proof that special susceptibility may exist in a family. A hard-worked Welsh clergyman had been accustomed to irregular meals, and had consumed in large quantities a cheap, strong tobacco. He

became nervous, and almost blind, and he recovered on giving up smoking. He told me subsequently that two of his brothers, farmers, had experienced precisely similar symptoms, and apparently in connection with the same cause. I did not see either of them, but I believe both were, like himself, almost wholly cured by abstinence from smoking. These two groups of cases, in each of which more than one member of the same family suffered from the same cause, bear to my mind very strong evidence in favor of the opinion that it is under the laws of idiosyncrasy that we must seek the explanation of chronic tobacco poisoning. It occurs to those in whom the nervous system, possibly the nervous tissue, has susceptibilities which are not shared alike by other members of the community. I have alluded to the fact that this susceptibility is not usually simple or immediate, as in the case of arsenic; it is almost always in those who have been long accustomed to smoking that these nerve phenomena occur, and they are probably indicative of a state approaching to saturation. If it be asked what are the other contributory causes which assist in producing the outbreak, I should be inclined to suggest that very varied influences which reduce tone may help; over-work and anxiety may assist, and so probably may the sudden loss of outdoor exercise. I do not think that the use of stimulants is often a contributory cause, at any rate, not unless to such excess as to interfere with appetite for food. Many facts which have come under my notice suggest that total abstainers from alcoholic beverages are, if they smoke freely, in much more danger of chronic tobacco-poisoning than those who drink them.

Respecting idiosyncrasies as regards *Arsenic*, I have to remark that they are not nearly so marked nor so common as those respecting some other drugs. I have met with exceedingly few cases in which small doses disagreed. It is very exceptional indeed that a patient complains of inconvenience at the outset of a course. Usually, symptoms occur after the drug has been to some extent pushed, and they for the most part necessitate its discontinuance or reduction. I am alluding now to the well-known symptoms of conjunctival irritation and pain in the stomach and bowels. Respecting these, I think, tolerance is never obtained, and if once they have occurred they are prone to occur again if the same doses

are given. In English practice, individual susceptibility to arsenic does not seem to vary much. Most persons, as I have said, can take it, and very few can take more than a certain dose. I have never myself seen a case in which it could be pushed beyond a certain point without definite disagreement. The most remarkable fact in reference to idiosyncrasy is its occasional production of the form of neuritis which we know as herpes zoster. It is now more than ten years since I published a series of cases illustrating the occurrence of herpes zoster in patients who were taking arsenic, and since then I have seen so many other examples of it that I cannot entertain the slightest doubt as to the direct connection between the two. The herpes eruption may occur in the forehead, face, or trunk, and it is wholly indistinguishable from the herpes in these regions which is not due to arsenic. It runs the usual course and disappears after its normal duration, whether the arsenic be continued or not. The patient does not, as a rule, show any constitutional symptoms of arsenic intolerance, and it is almost invariably the case that he has taken the drug for some weeks before the symptom appears. The advent may now and then be delayed until a week or ten days after the course has been suspended. A precisely similar interval is sometimes seen in cases of sympathetic ophthalmitis, in which affection—probably, also, a form of neuritis—the inflammation of the second eye may begin to develop a week or ten days after the entire removal, by excision, of its cause. These facts would suggest that the neuritis is in each instance slow in its stages, and that, having been once initiated, its local processes cannot be stopped. The number of those who show arsenical herpes is very small in proportion to those who escape, and, if it is to occur at all, I think it always happens during a first course. I have never seen it in a patient who had been accustomed to the drug for a long time. Thus, its occurrence must certainly rank as the result of an idiosyncrasy; and this suggestion opens the way for the further one—that we ought, perhaps, always to regard herpes zoster as, in the main, an indication of personal peculiarity. It certainly does not appear that all persons are equally liable to it, since it happens to but very few, and is never associated with any special form of ill-health. Excepting the single fact, just alluded to, of its occasional production by arsenic, we know of no causes which

can evoke it. I have seen arsenic produce herpes in two different regions at the same time, but I do not remember to have ever witnessed a second attack in the same individual. In reference to this, however, it is but fair to add that I have usually carefully avoided this drug after an attack of shingles, since the latter is often a very serious trouble. A few persons —a very few—are liable to the occurrence of an eruption of herpetic vesicles over and over again in the same part of the skin, and, as is well known, examples of recurring herpes on mucous membrane are common, as, for instance, on the lips and genitals. Sometimes recurrences take place once a month, or even oftener, the eruptions always passing through the usual stages, and undergoing a speedy and spontaneous cure. There is never any peculiarity of health to be made out in explanation of these cases, and they must, I suppose, rank as idiosyncrasies. Homœopathists are welcome to make what use they can of the fact that arsenic will often cure these cases and prevent the recurrence. I have repeatedly seen herpes of this kind remain in abeyance for many months while the patient was taking arsenic, and show itself again when the drug was suspended. I have seen one case which to some extent offers an exception to some of the statements which I have just made, inasmuch as the patient was liable repeatedly to an eruption from arsenic. The eruption always came soon after beginning the remedy, and he was well accustomed to its use. The patient is a gentleman now near to sixty, who has suffered from psoriasis most of his life, and for whom in early life Sir Benjamin Brodie prescribed arsenic. He has often taken it since, and its usual effect is as stated. On one occasion he took it in order to show me the character of the eruption produced. It had been called "eczema," and, although it certainly had the local features of an erythematous eczema, yet it occurred, like herpes, unsymmetrically on one arm in large patches, which disappeared spontaneously after a few days.

LECTURE III.

MR. PRESIDENT AND GENTLEMEN,—I hope I have made it sufficiently clear that I am not purposing to trouble you with any list of idiosyncrasies. As well for your relief as my own exculpation, it is perhaps desirable that I should again make this statement. What I said about egg-poisoning in my last lecture was intended to suffice as an illustration of what is possible, indeed I would suggest not infrequent, in reference to a whole group of food-idiosyncrasies. The same remark applies to the somewhat detailed discussion on the susceptibilities in reference to tobacco. We are engaged in the examination of the laws of idiosyncrasy, and not in any attempt to record exhaustively its facts. I shall bring before you no more narratives of cases or statements as to facts than may seem absolutely necessary to illustrate the general laws to which we refer. In Monday's lecture we glanced briefly at certain peculiarities of structure which are sometimes met with in external parts which are both congenital and hereditary. For these I proposed the name of structural idiosyncrasies, and asked that we should apply the knowledge that we can easily gain respecting them to those which are much more obscure, from the fact that they are betrayed only by derangement of function.

I next alluded to certain forms of inflammatory disease not present at birth, and in most cases not commencing until a considerable period after it, but concerning which the general facts suggest that they must be due to some inborn peculiarity of the structure implicated. Lastly, I adverted to examples of complicated or secondary idiosyncrasy in which the individual peculiarity is not displayed immediately on the application of its exciting cause, but waits, as it were, for the co-operation of other influences. We found one of our best illustrations of this in the chronic poisoning, denoted chiefly by amblyopia, to which smokers are in exceptionable instances

liable. I concluded my lecture by a brief allusion to idiosyncrasies in reference to arsenic, and was obliged by want of time to defer till to-day what I have to bring before you on the important subject of the iodides and similar salts. I shall be compelled to omit altogether what I had prepared in reference to belladonna, opium, and certain other drugs, an omission which I shall regret the less because, as just said, I am seeking only for illustrations of law, and of these perhaps we shall have enough.

The iodides and bromides offer us, indeed, such excellent and various illustrations of the laws of idiosyncrasy that I am almost tempted to wish that I had excluded all others, and thus secured more time for the consideration of them. The modes in which these salts can produce disagreement are exceedingly varied, and concern very different parts of the system, while many of them are displayed by the skin in a manner which permits of their easy identification and examination. Not even the syphilitic virus itself is capable of producing a greater multiciplicity of pathological changes in the integument. Now it is a hæmorrhage, now a thrombosis, now an erythema, now a bulla; the commonest of all forms is an acne, but in addition to it and those which I have named we may have at the same time pustules, button-like tubercles, and in aggravated cases enormous soft tuberous growths.

If time had permitted me I should have taken occasion to advert also to a topic of much importance to us in operative practice, and should have suggested that in a considerable number of the accidents with anæsthetics it is idiosyncrasy which we encounter. Many of the deaths from chloroform occur to persons apparently in perfect health, and when only small quantities have been given and with every possible precaution. Such cases ought probably to be placed side by side with those in which individual susceptibility becomes unexpectedly revealed to us in reference to such drugs as opium, belladonna, and the like. We waste our labor when we search for proof of heart disease, or some flaw in the apparatus, or some want of care in the administration. The last patient in whom, under my own care, we came near to a fatal event with chloroform, was a man at Moorfields, in whose case extraction of cataract was about to be performed. He would certainly have died but for the vigorous use of artificial res-

piration and the nitrite of amyl. In discussing the case afterwards I was reminded by our house-surgeon that three years before, when the other eye was operated on, the same man had caused us alarm by precisely similar symptoms. The occurrence had unfortunately been forgotten. It is probable that there are persons in whom, owing to peculiarity of nerve organization, and not in the least to anything which we can recognize as ill health, the inhalation of chloroform will always cause an extreme depression of the heart's action. In such, if the precaution of giving beforehand a dose of opium, alcohol, or ether, be omitted, there is the utmost risk of a fatal event. Probably each anæsthetic has its related conditions of idiosyncrasy.

I have passed over also another subject, owing to want of time, which has considerable surgical interest. It is the influence of inherited structural peculiarities in favoring the occurrence of lesions which at first sight suggest no suspicion of such origin. Examples we have of this in the easy occurrence of dislocation, owing to peculiarity in joint formation; and the proneness to intussusception of the bowels, owing to the cæcum and ascending colon being provided with a mesentery. I have several times known the tendency to intussusception show itself in several members of the same family, and in one of the most marked instances in which several of the children had died of it in succession the condition referred to was found at the autopsy of one of them.

I now turn to the subject of the idiosyncrasies in reference to the iodides and bromides. It seems probable that the difference in form which the inflammation of the skin assumes is dependent upon the peculiarities of the individual, for if the patient has shown a tendency to an unusual development of the iodide rash, on one occasion, and the same salt be given to him on another, even after a long interval of time, it will produce exactly the same effect. This I have witnessed repeatedly. Idiosyncrasies as regards the iodide are not manifested solely by the skin, are perhaps not primary in it, and are seldom confined to it. Congestions, with excessive secretion, of certain tracts of mucous membrane, are of yet more frequent occurrence than are skin rashes, but they differ from the latter in that they are, in a majority of instances, only transitory. An eruption on the skin, once developed by the

iodide, persists—and indeed becomes increased—so long as the drug is given; sometimes, even after it has been suspended. The iodide coryza, as it is called, on the contrary, seldom lasts more than a few days; or if it becomes chronic, always subsides into a slight affair. Thus the one would appear to imply idiosyncrasy in a far less degree than the other, and this suspicion is borne out by the observation that in those who suffer from any of the more serious forms of skin eruptions, there are usually other evidences that the drug is producing injurious effects. The tone of the nervous system is always lowered by it, and sometimes to an alarming extent. There is, perhaps, no drug that can be mentioned respecting which idiosyncrasy is so common and variations in its degree so great. I have repeatedly seen doses of only half a grain produce, in the course of a couple of days, definite symptoms of poisoning; and, on the other hand, I have known a patient swallow an ounce and a half a day of the best Apothecaries' Hall iodide for a week together without any special effect, except a feeling of lassitude and want of tone. It is also very remarkable how, in a large majority of cases in which definite and peculiar susceptibility is at first manifested, tolerance may subsequently be attained, and doses of ten, twenty, thirty times the amount given without obvious effect. Probably there is no drug respecting which the assertion can be more easily demonstrated, that it is not the dose, but its effect, which we should regard. It by no means follows that because a patient has an idiosyncrasy against some drug or article of diet, that we ought wholly to abstain from its use. Rather this susceptibility, in the majority of cases, proves that in him minute doses will effect the cure first as efficiently as larger ones in others. I have repeatedly cured tertiary ulcerations of the throat and skin in patients in whom I was assured iodide was a poison in all doses, by giving it in quantities of half, or even a third, of a grain. In the year 1862, M. Bazin, of Paris, an excellent specialist, and at the same time an excellent physician, described a new disease under the name of *Hydroa.* Of this malady he recognized several varieties, one like varicella, one resembling smallpox. On reading M. Bazin's descriptions I was at once struck by the closeness with which they applied to several cases which had been under my own care, and in 1870 I wrote in the "British Medical Journal" a report on "hy-

droa," quoting M. Bazin's descriptions, and recording my own cases together with others supplied to me by my friends. I had previously been to Paris and seen M. Bazin's models. At that time neither of us had any idea of the usual cause of the eruption, nor, I believe, had any of our readers or critics. One of the latter suggested that we had not named it correctly, and that it was only an "urticaria bullosa," but in doing this he gave us no clue to its cause. At this date we all knew of the acne which so commonly results from the iodide, but were not aware that this salt could in connection with idiosyncrasy produce other symptoms so much more formidable. In the session of 1869, Dr. Cholmeley brought before the Clinical Society an example of a remarkable confluent eruption produced by the bromide; several papers had also appeared in Paris on the subject, and subsequently I convinced myself that almost all the examples of the disease to which we had given a new name were really the bullous form of the iodide eruption. From that time to this I have seen but very few examples of "hydroa," except as the consequence of iodides or bromides. It will be seen that we had made no error in observation, but had simply described and named a disease which was new to us, and new to medical literature, but of which at the time we had not discovered the cause. When the cause was found the name became of little importance. As already hinted, however, we have by no means in mentioning the iodide acne, the iodide "hydroa," concluded the list of symptoms due to this drug. Respecting a case which I have to mention in the next group, I shall not be surprised if many remain still in doubt as to whether it ought to be placed in this position. A man came to me at the London Hospital covered with large soft tumors, some of them as big as a child's fist. They occurred chiefly on his face, chest, and limbs. That I may avoid a lengthy description, I will simply refer you to the sketch which hangs on the wall, which is a rough copy from an excellent drawing which I now display. It will be enough to say that the moment I saw it I believed it to be an iodide eruption, and that my diagnosis was based upon the recollection of other cases, unquestionably due to that cause, which I had previously seen. The man had just been discharged from another hospital, and on inquiry I found that during his month's stay in it he had been steadily taking the iodide in increasing doses

the whole time. The eruption was not present in the least before the drug was begun. The patient died within a fortnight of his admission under my care, from exhaustion, apparently due to the extent of surface implicated. The post mortem revealed nothing which threw further light upon the cause of his malady. It seemed probable that he had never had syphilis. He had gone into the hospital on account of another complaint, and when the eruption appeared it was considered to be syphilitic, and instead of suspending the remedy, its dose was increased. The dose was, however, never more than fifteen grains three times a day. To those who are still inclined to doubt whether this terrible eruption was really due to the iodide—and I admit I have no right to be surprised if the number comprises all my audience—I would put the question, What else could it be? The microscope showed in the tubers only the results of common inflammation, and there was nothing in the least suggestive of malignancy. Above all, I would ask that a careful comparison be made with the other drawings from another case which I have placed by its side. It will be seen that the two closely resemble each other, and differ only in the greater severity of the one I have just described. In all essential features of the eruption they are exactly alike. Now there is not room for the slightest doubt as to the cause of the eruption in the second case. It was produced by the bromide of potassium, and it occurred in connection with that drug twice to the same child. I may here remark that I know of no difference between the eruption due to iodides and those due to bromides. The same variety occurs, I think, in both, and the general laws as to idiosyncrasy are the same. The iodide, however, depresses the system far more than the bromide. The eruptions produced by bromides are, I think, attended by less constitutional disturbance, even when locally very severe, than are those due to iodides. A case well illustrating this came under my notice a few weeks ago. I was asked by a medical friend to see an infant in whose case the diagnosis had first been varicella, and next smallpox. Complete isolation had been enforced, but as the eruption had steadily got worse, and the child now at the end of a month remained perfectly well as to health, all had become convinced that there was a mistake. I have mentioned the names which had been given to the eruption as the best

means of suggesting the appearances it at first assumed, and I may add that the surgeon who attended the case was an experienced and sagacious man. The infant, when I saw it, was fat and smiling, but with its face and all four extremities absolutely covered with large buttons of most formidable appearance. Many of them had scabs, but none of them had ulcerated much. At first I was told that the eruption was present before a medical man was consulted, and before any medicine had been given, but on pressing for accurate dates, I found that my suspicion was correct, and that the surgeon was first consulted for a slight feverish cold, for which he prescribed small doses of bromide of potassium, and that the rash did not begin to appear till three days later. No suspicion having been entertained, the bromide had been continued until the day of my visit. My friend told me that he was very fond of the bromide for children, and had given it in thousands of cases, but had never before seen any eruption produced. In looking back on the experience of former years, I recollect several cases under my own care, and that of other surgeons, in whom I think there is great reason to suspect that persistence in the iodide caused death. They were cases in which an eruption which was probably due to it alone was mistaken for a specific one, and the drug pushed still further. In the report which I published in the Clinical Society's "Transactions" I have published two cases in which our eyes were opened only just in time. They were all cases in which the salt had been continued for some weeks. It may be possible, however, for fatal poisoning to occur in connection with the first few doses. In the case of a man admitted into the London Hospital some years ago, only two or three doses had been taken, and an eruption which looked like hæmorrhagic variola had been produced, together with such an amount of swelling about the neck and throat that nothing but the prompt performance of tracheotomy could have averted immediate death. The facts respecting this case were published by my colleague, Dr. Stephen Mackenzie, and the same indefatigable observer has also placed upon record the most extraordinary example of iodide poisoning with which I am acquainted. In this instance, an infant, aged five months, died of iodic purpura after a single dose of only two grains and a half.

The domain of idiosyncrasy in respect to Specific Fevers is exceedingly important as regards both the physician and the surgeon, and I shall make no attempt to distinguish arbitrarily between their interest in this matter. That which the surgeon is familiar with in the case of syphilis, and those remote and exceptional sequelæ of measles, scarlatina and smallpox which come under his care, may be used to throw light upon the facts which fall under the observation of the physician in the earlier stages of these latter maladies. There is probably no good reason for believing that the specific animal poisons can vary in their intensity beyond certain narrow limits. Different epidemics of scarlet fever and smallpox do unquestionably vary somewhat, but after all, the limits of deviation from average intensity observed in different epidemics are as nothing compared with what we witness in different individuals attacked in the same epidemic and apparently under like circumstances. In the latter case we see constantly that it is possible for two individuals infected from the same source and with the same virus to show, the one an eruption consisting of only a few spots almost unattended by fever, while the other developes a confluent or even hæmorrhagic exanthem, which will prove fatal in a few days. Is it possible to give any explanation of these individual differences, except by attributing them to individual peculiarities, and when these peculiarities arise to an extreme height, can we name them more conveniently than as idiosyncrasies? I will endeavor to illustrate the subject by facts which have come under my own personal cognizance. The portraits which I produce exhibit a gangrenous and ulcerative eruption which occurs in connection with varicella. They are, in fact, a peculiar form of varicella in which the eruption tends to persist and the individual vesicles ulcerate or become gangrenous. The evidence upon which I venture this unqualified assertion is, I think, conclusive, for this eruption has been observed repeatedly, with the history that in the first instance it was supposed to be chicken pox, and while chicken-pox was in the family; and in a few yet more definite cases, the surgeon who identified the gangrenous case was the witness at the same time of varicella in its ordinary mild form in the patient's brothers and sisters. The facts have fallen under the observation not of myself alone, but of many others, and they receive important incidental cor-

roboration from several narratives published long ago, and without full perception of the interpretation of the facts. My present object is to cite them simply in proof of the possible influence of idiosyncrasy in modifying the course of an exanthem. The case to which the portrait which I next show belongs, was that of an infant to all appearance in perfect health, who had several brothers and sisters, all healthy, who had all been vaccinated at the usual time with the usual results. In this child, however, vaccination was followed on the seventh day by a general eruption on the skin, which on the tenth day looked like ordinary smallpox, which subsequently became gangrenous, and caused death at the end of a fortnight. The state of the child's skin at the time of its death is well shown in the portraits.[1] It will be seen to be covered by spots and patches of gangrene, the sphacelus involving the whole thickness of the skin. There is no form of variola known which in the least resembles this condition, and none will suggest, I think, that diagnosis. On the other hand, there is a well-known eruption, usually slight and mild, but occasionally very definite, which occurs after vaccination, to which the term vaccinia exanthem is appropriate. My suggestion is that this eruption bears the same relation to the ordinary mild vaccinia rash which gangrenous varicella does to the common type of chicken-pox, and that its exceptional severity was due to individual idiosyncrasy.

I show next another portrait from a similar case which was observed by Mr. William Stokes in Dublin, to whose kindness I am indebted for the portrait. The general facts were similar to those in my own case, with the difference that the eruption occurred a day or two earlier after vaccination, that the patches of gangrene were more irregular and more superficial, and that the child survived. I do not know that any other examples of gangrenous vaccinia have as yet been published, but I have no doubt that such will in the future be observed. I have now related some extreme instances illustrating the influence of idiosyncrasy in causing severity of exanthems which are usually very mild. Probably there are quite as striking examples of the converse, instances in which the

[1] One of these is copied to illustrate my paper on this subject in this year's volume of the "Medico-Chirurgical Transactions."

specific animal poisons pass through the system and produce little or no obvious effect. It may be that these are much more common, and that they go far to explain some of the great apparent irregularities in the course of the exanthemata. We often meet with individuals who tell us, for instance, that they have never had scarlet fever or measles, although they were exposed in childhood at the same time that their brothers and sisters suffered; and we often see these diseases go through a family and leave one or more children apparently untouched. Still oftener will it happen that one or several individuals under such circumstances will show only a very slight febrile ailment, without any of the more characteristic symptoms of the disease, while others suffer severely.

It has happened to me recently to witness a family epidemic of scarlatina which remarkably illustrates what I wish to say. In mentioning its details, let it not be supposed that I think that it illustrates anything unusual; on the contrary, I believe that similar occurrences fall very frequently within the experience of those engaged in general practice.

My facts are briefly these: a child was affected with sore-throat and feverishness, but had no rash whatever, and beyond a slight fur, no change in the condition of the tongue. She was well in a day or two, and nothing subsequently occurred to her. Four days later another child sickened with exactly similar symptoms, but more marked, and in her the usual rash of scarlatina soon appeared; and subsequently a little boy went through the same course of events. Both of these observed precisely the same periods as regards the progress of the disease; both had nose-bleeding on the same day, followed by rheumatism beginning in the same joints, the wrists, and both had great swelling of the cervical lymphatic glands. Subsequently an older sister, who was believed to have had it some years ago, went through a rather sharp attack. Thus, I take it, there can be no doubt that the disease in this instance was scarlet fever; but to prevent any question on the score of diagnosis I may add that one of the patients was seen by Dr. Gee.

It is to what I have next to state that the chief interest attaches. The child who was the first ill, and who had but a day's sore-throat, no tongue affection, no rash, no peeling, was subsequently most fully exposed to the risk of infection, and

did not take it, conclusively proving, I think, that the trivial illness she experienced was the fully developed malady so far as her idiosyncrasy permitted it to show itself. But I have other facts yet more curious. Almost the whole family, and it was a large one, suffered one after the other, during the two or three weeks that the disease was in the house, from sore-throats and a day or two of feverishness. Several of them had never had scarlet fever before, but as to some others, it was doubtful. In all, the cervical lymphatics swelled more or less, but in no instance was there the slightest peeling or any sort of sequela. I have yet to add that, when the first child had sore-throat, I was at once told that the family of a cottager living at a short distance from the house had just been suffering from similar throats. This cottager was the milkman, and there had also been personal communication between the two houses. On inquiring at this cottage, I found that the father, mother, and two grown-up daughters had all in succession had sore throats, attended for a day or two with rather sharp febrile illness, but in no single instance had any rash been observed, nor had any peeling of the skin followed. They were not known to have had scarlet fever on any former occasion, and laughed at the suggestion that what they had now had was it. It may be that after all it was not, but the dates fitted well with the possibility that the first of the children affected had got the contagion from this source. The inference which I wish to draw from such narratives as this, is that it is possible for the individual peculiarity of patients —a peculiarity which may be hereditary and may affect many individuals in the same family—to so far modify and minify the course of an exanthematic fever as to make its recognition quite impossible, and that we ought to seek for the explanation of these remarkable differences, amounting in many instances to almost total suppression, not in variableness in the degree of epidemic virulence, nor in the hygienic surroundings, nor in anything which can properly be called the state of health of the patient, but in that of which we can give no other explanation than that it must rank as an idiosyncrasy.

It is needless to point out what a pitfall of fallacy is here opened for the feet of the investigator. How easily may we find ourselves attributing to influences which were wholly inoperative, results which must simply be put aside as those

which we ought to have expected under the known laws of idiosyncrasy. I will leave for the present the examination of those laws as they influence the course of a certain other specific fever. This is the time at which I ought properly to mention the facts which, in the case of the protracted fever known as syphilis, illustrate its extreme inequality of incidence in different persons. I must not, however, enter into any details now, and I leave them aside the more willingly because the facts to which I refer are matters of universal knowledge. We all know how differently chancres behave in different persons; how they may become phagedænic or sloughing without our being able to assign any cause for the peculiarity. We know, also, that the syphilitic rash in many cases is wholly omitted, together with the rest of the train of secondary phenomena. We know also how, when secondary symptoms do appear, how infinitely variable they may be in form and in severity. A roseola, a lichen, a psoriasis, a rupia, an eruption indistinguishable from variola—such are a few of the forms which we frequently see. Impressed by these startling differences, good observers have tried to make out that there are different kinds of virus, but all the facts are in opposition to such hypotheses. I must content myself by suggesting that they are due to inherent differences in the individuals who receive the virus, and must now pass on to other topics.

Let me next remark that, not only is the sphere of individual peculiarity almost unbounded in its power of modifying the force of the primary outbreak of an exanthem, but that it exercises a great influence in determining who shall and who shall not, suffer from complications and sequelæ. Under this latter term I am alluding rather to what may be called accidental sequelæ; the inflammations of various organs and parts which occur exceptionally and at some little distance of time, and which often brings the patient under the care of the surgeon. The peeling of the skin, and possibly also the nephritic desquamation which follows scarlatina, are sometimes classed as sequelæ, but are really parts of the primary disease. As I think my narrative has conclusively shown, peeling of the skin is not a sequel of scarlet fever *per se*, but of one of the symptoms of scarlet fever, namely, of *dermatitis;* to which it is always proportional, and of which it is the necessary consequence. Inflammations of the ears, and of the bones or joints,

stand, however, in a different category, and so does the ophthalmia tarsi, which often follows measles, and the pruriginous affections of the skin which are not infrequent after both vaccinia and varicella. When these events happen, they occur probably not by any means as necessary parts of the disease, but rather as revealing idiosyncrasy in the individual. Inflammations of the ear and allied affections may possibly in some instances reveal diathesis rather than idiosyncrasy. That is, it may be that they occur because the patient is distinctly scrofulous, or the subject of some other form of recognized ill-health. Even as regards them, however, I much doubt whether this is always true, and respecting the affections of the skin to which I have referred, I am sure that they often occur to those who are in good health, and that they are proof of idiosyncrasy only.

Differences in the degree of irritability of the skin are common, and may proceed to great length. Flea-bites, woollen clothing, nettle-stings, and the like, produce on different persons extremely different results. Urticaria is a form of dermatitis of which probably but few persons are susceptible, and in those in whom it occurs it demonstrates or reveals this susceptibility and nothing more. The patient who gets nettle-rash whenever he eats lobster may yet be able to digest lobster with as little inconvenience as other people. It is his skin, or perhaps the nerves of his skin, in which the susceptibility alone lies. In this suggestion rests, so far as I can see, the real explanation of the maladies which have been grouped together under the name of *Prurigo*. There is no one disease to which that name is applicable. What we witness is the power of various causes of local irritation to provoke pruriginous dermatitis in those in whom idiosyncratic susceptibility exists. Now it is the presence of pediculi which is the starting-point; in other cases of fleas; in yet others the occurrence of an exanthem, such as varicella; while in all, the morbid condition, once initiated, is kept up by the patient's unwise efforts to relieve it, and by the mere fact of its having got possession. I repeat, there is no such disease as "Prurigo," a malady which has been described by a high authority as if it were self-existent, and alike causeless and cureless; but there are plenty of persons born with that peculiar irritability

of skin in which a variety of causes may evoke the symptoms to which that name has been given.

We may probably, with much confidence, place diphtheria and erysipelas side by side as diseases,—or rather type forms of the inflammatory process,—which obey parallel laws. Probably no human being is exempt from the liability to them, but at the same time it seems certain that the degree of proneness varies very much in different individuals, is persistent through life, prevails in families, and is matter of inheritance. Thus, then, we assert for both the existence not unfrequently of a sort of constitutional predisposition to which the term diathesis, or even in some instances, that of idiosyncrasy, is applicable. Both may become contagious, and of both exposure to direct contagion is probably by far the most efficient mode of production. Many persons will take them by contagion in whom other causes would have proved abortive. But both are capable of spontaneous origin or, to speak more correctly, may be produced by exposure to certain non-specific influences, such as the ordinary causes of catarrh, injuries, sewer gas, and some others. It is most important that all who value accurate classification should deny both as regards erysipelas and diphtheria their right to rank with those fevers which are due to specific animal poisons. With these, as it seems to me, neither of them has any real affinities. In both, I repeat, that with which we have to deal is a personal and inherited peculiarity, giving proclivity, when certain exciting causes are brought to bear, to peculiar forms of inflammatory action. In both the disease is probably local at its origin; in both it spreads locally by the contagion of continuity of tissue, and in both constitutional symptoms result which are in some ratio with the extent and severity of the local action. Neither of them in the least protects the individual from future attacks; indeed, and although in each single attacks are common enough, yet it is generally acknowledged that those who have suffered once are more prone than others to suffer again, and in both there are not infrequent instances of proclivity lasting through many years.

In erysipelas the essential feature is a spreading inflammation of lymphatic spaces in the skin producing great œdema, and usually great enlargement of blood-vessels. In diphtheria we have a spreading inflammation of a mucous membrane,

attended by œdema, dilatation of blood-vessels, and the formation, in greater or less quantity, of a fibrinous pellicle. Although I mention skin as the seat of the one and mucous membrane of the other, let it be distinctly understood that these are only the most usual sites; both may attack cellular tissue or wounds, and each may occasionally affect the usual territory of the other.

There may be some doubt as to how, when these inflammations affect repeatedly the same individual, the fact should be explained. It may be that one attack leaves a really increased proclivity to a second, or it may be that the first simply revealed a proclivity which had existed all along, and of which the later ones are merely the manifestations without addition. Probably both explanations are true.

It is, I presume, not necessary that I should produce before this audience any citation of evidence in proof that a personal liability to erysipelas and diphtheria may be witnessed. All are familiar with them. It is probable that there are some slightly marked affinities with the diseases just named and such maladies as carbuncle, boils, and acne. Respecting all these we have the same questions to ask, how far are they dependent upon constitutional causes? and how far upon those which are local? We may take it as certain that in all there is a double causation, and our task is to assign in each instance the relative share of each. In the first place we may say that the liability to acne, to boils, to carbuncles, is often a family matter, and is seen to occur with unusual frequency in relatives. It is often also a personal matter, as is proved by its persistence or recurrence at various periods of life. Further, the association of these maladies with more or less temporary disturbances of health, with diabetic dyspepsia, with constipation and defective tone, is a matter of constant observation.

Whoever has reflected, however, as to the real nature of carbuncular inflammation, will, I think, see reason to believe that its peculiarities are to a large extent local ones. Respecting both boils and carbuncles, it is the fact that the most efficient measures of treatment are those which appeal to the local condition. This is true of all the different measures in repute, however dissimilar at first sight they may appear. Some have for their object the protection of the inflamed area

from further irritation, as when we cover up a boil or commencing carbuncle with leather plaster, others the subdual of the inflammation by means of cold and the like, as when we use an ice-bladder or a strong spirit lotion. Nor do those who adopt a time-honored practice of an early free incision depart from this principle, for their object is still the mitigation of local inflammation by the relief of tension, and by permitting the escape of contaminating matters. From acne, impetigo, and ecthyma to boils is in many cases a matter of degree, and that there is little or no real distinction between a large boil and a small carbuncle, all will, I think, admit. The age of the patient, and his special diathetic condition at the time, their size, the influence of different modes of local treatment, are the conditions which determine the final result. If a boil occur in an elderly person, on the nape of the neck, where it is likely to be irritated by the shirt-collar, and especially if, instead of being symmetrically protected from the first, it be further bullied by premature squeezing and the like, it is very likely to pass on into a carbuncle. I claim the strong testimony which has been borne by Sir James Paget and others to the efficiency of simply protective measures in preventing the development of small carbuncles, and obviating the supposed necessity for incision, as proof that local spreading is, in the main, due to local causes, and has no necessary connection with the general health. In other words, it is not a thing which is inevitable. On the other hand, we know of no internal remedy which possesses any marked power in arresting carbuncular inflammation.

If now we ask, In what does the peculiarity of carbuncular inflammation consist? in what does a carbuncle differ from a boil? I should be inclined to reply that it is solely in this tendency to spread. The spreading is effected by contagion of continuity, and it has certain marked resemblances to what we observe in erysipelas. A carbuncle might almost be defined to be *an erysipelatous boil* with the proviso that the expression is not intended to imply identity of inflammatory type, but only close similarity. Throughout its whole course, and however extensive it may be, a carbuncle, in combination with its erysipelatous method of spreading, contains the characteristics of the furuncular process. Its secretions and its core, or slough, are exactly like those of a boil, excepting that

they are much more abundant. In a general way there appears to be a natural tendency, independently of treatment, to the arrest of the process, and this is not generally observed in erysipelas. In other words, erysipelatous inflammation is more intensely infective than is carbuncular. I must, however, qualify even this admission of difference by reminding you, that carbuncular inflammation has such an injurious influence on health, that in all cases in which its spread continues in spite of treatment the patient quickly dies. Thus many cases occur in which no proof is given of tendency to arrest. It may easily be the fact that in some states of system, and some types of carbuncle, there is as little tendency to arrest as there is in phlegmonous erysipelas.

I fear I have wandered rather far from the subject of diathesis, but my argument, if I have succeeded in making it clear, is this: that there exists a certain state of tissue and constitution, different in each instance, which gives proclivity to diphtheria, to erysipelas, to acne, to boils, and to carbuncle, and that while the importance, the almost paramount importance in many instances, of local influences in exciting and aggravating is kept in mind, the diathesis in the background must not be forgotten. Further I have argued that carbuncle is the result of a sort of combination of the erysipelatous and furuncular types of inflammation. I feel sure that these views as to pathology will be found to fit well with therapeutics.

Tetanus is a malady so rare, that if we admit its possible spontaneous production without regard to previously existing proclivity, we must not expect to find many instances in proof that the latter does sometimes exist. Yet there are such, and possibly, if looked for, more than we might suppose. The influence of race is proved by the great liability both of infants and adults among the negroes, and also of certain animals. I have met with a few cases in English practice which appeared to imply that there might be an inherited proclivity to tetanus. A man who looked quite well walked into my room as an out patient at the Metropolitan Free Hospital and gave me his own diagnosis by declaring "I am going to have lockjaw." I found that he had recently had a slight wound on one hand, and that he was beginning to feel some stiffness of his jaws. His symptoms, however, were exceedingly slight, and his anxiety was caused by the fact that his father had died of

tetanus. The poor fellow's foreboding was rapidly realized, and he died within a week.

Here, for the present, Mr. President, I must take leave of the subject of idiosyncrasy. I have endeavored to claim for these forms of individual peculiarity a much wider sphere of influence than is usually accorded to them. It is necessary, I am well aware, that one should exercise some scepticism in accepting doctrines of the kind which I have brought before you. To fall back on the suggestion of an idiosyncrasy is so easy that it may easily become the resort of intellectual sloth. We must not, however, be deterred by this consideration from the acceptance of such doctrines if they are true. That they are so within certain limits I cannot doubt, and the problem before us is to find those limits. If we fail to recognize, or if we forget, the influences of idiosyncrasy, we shall not only waste much time in our processes of clinical research, but we shall be in constant danger of coming to wrong conclusions by declining to accept evidence as to cause which is really sound, and of adopting false principles in reference to treatment. In every example of a curious and unexpected form of disease, our minds should, I think, first ask the question, "How much of this may possibly be due to the individual peculiarities of its subject?" We are often on a wrong track if we seek to make external influences explain the whole.

Let me add, in conclusion, and it seems to me of great practical moment, that the individual peculiarities of all patients should be carefully studied, and that they should themselves be made intelligently acquainted with them. There are few of us without our idiosyncrasies, and their variety is innumerable. If it should become the custom for parents to record some sort of life-history of their children in permanent form from the birth or infancy onwards, noting all the peculiarities in an individual by the aid of medical observation, not only would much be done in the way of preventing subsequent errors in the treatment of disease, but valuable contributions would be made to our knowledge of its real nature. It is not only as regards the prescription of our drugs that a knowledge of our patient's peculiarities becomes important to use in practice. The advice we have to give in respect to places of residence, mode of life, and of general management are often of far more importance than the medicine prescribed. Its wisdom or the

reverse may depend on our knowledge or ignorance of the individual peculiarities, and those peculiarities frequently do not display themselves in any of the existing symptoms, but can be recognized and revealed only by a correctly kept life-history.

LECTURE IV.

Mr. President and Gentlemen,—We turn to-day to a new department of our subject. I have been dealing hitherto with fixed inborn peculiarities which are for the most part beyond our explanation, but in consequence of which disease in individuals may assume very peculiar features. How peculiar and exceptional those features may be I have illustrated by the production of cases of bullous, pruriginous, and even gangrenous varicella, and of two examples of gangrenous vaccinia. I have also reminded you of the great differences in different individuals in the type and severity of syphilis, and I have shown, too, how, not only may the exanthemata sometimes attain most unusual severity, but that not unfrequently all their usual symptoms may seem to be suppressed and the patient may pass through a complete attack scarcely knowing that he has been ill. We have further glanced at the facts which prove that the iodides and bromides, remedies in daily and most frequent use, and for the most part very harmless, may occasionally act as violent poisons. Lastly, I have urged that in many other morbid processes originating in connection with ordinary and known causes, or it may be from local infection, the peculiar constitution of the individual takes an important share as well in increasing the proclivity as in modifying the result. Instances of this I found in such diseases as diphtheria, erysipelas, and carbuncle. Respecting these and the other examples of personal proclivity or immunity which I have ventured to cite, I have maintained the same argument, that they are all independent of everything that is to be recognized in the state of health of the patient, and that they imply that which is conveniently known as idiosyncrasy. Much, indeed almost all, that I have advanced is, I am well aware, by no means novel. I have been dealing with old names and well-established doctrines, and my reason for bringing them before you has been, not so much the hope of

being able to contribute new facts or theories of my own, as the desire to set in better order that floating knowledge which is a general possession. In the lectures which are yet to come I shall restrict myself to the same design. I am going to ask your attention to a great number of topics, to many of which I shall advert with such brevity that it would not be possible for me to enforce original views even if I possessed them. The bond of connection which will associate these various subjects will be the endeavor to discover whether there exist any large groups of states of personal peculiarity giving special proclivity to diseases which can be suitably defined and named. If the reply to this question be in the affirmative—if we see reason for believing that those who become our patients do really differ one from another in respect to definite tendencies, which are of prolonged duration and upon the knowledge of which we can conveniently base not only our prognosis but our treatment, it will next be our duty to examine as to the real nature of those states and the laws under which they came into being and continue to exist. I have dismissed the consideration of temperaments as being when correctly limited an unproductive field of research, but in doing this I expressed a belief that the investigation of the diatheses stood in a very different position. In the one we deal with the peculiarities of race and the results of what may be termed breeding, in the latter we begin much nearer home and investigate the consequences of a variety of influences which are capable either separately or together of producing chronic disease. For, in fact, what we mean by a diathesis is little other than an exceedingly chronic disease. It is a disease or taint which lasts a lifetime, which may be active at times, and latent at times, and which may be handed on to another generation. If we accept as a definition of diathesis that it is *any condition of prolonged peculiarity of health giving proclivity to definite forms of disease,* we must admit at once that it will comprise groups of maladies which are very different in their nature. Of some of these states the causes are very definite and specific, while of others they are much the reverse. Of those which are most definite, such as leprosy and syphilis, it may perhaps almost be doubted whether there is any convenience in calling them diatheses at all. In certain stages when the disease is active there is assuredly no object in

using any other word than the substantive name. It is chiefly when external symptoms are absent for a time in the individual, or when we witness the phenomena of hereditary transmission, that we feel to need the addition of such a word as diathesis. It is in most conditions easy to say of an individual "he has got syphilis," or "he has not got syphilis," or "he is the subject of leprosy;" for the disease in each instance is a definite one, and there is no such thing as a state of physical proclivity to either. Such maladies, however prolonged may be their possible duration, stand in the very different category to scrofula, rheumatism, or catarrh. These latter are diatheses which may vary within the widest limits in the individual, and are so common as to be well-nigh universal. We can scarcely assert of any one that he is free from them, and the question is rather as to estimation of the degree. They are diatheses in the truest sense of the word, and they belong to the whole human family, and possibly, indeed, to all vertebrate animals. The more important questions to be put in reference to them are not, have you got rheumatism, scrofula, catarrh, etc.? but rather, in what degree are you catarrhal, rheumatic, or scrofulous? The degree of susceptibility in each varies exceedingly in different persons, and in many instances rises to such a height that it constitutes a very serious disease. To these, so to speak, universal diatheses I shall have to recur in my last lecture.

In the same sense that we seek to know respecting our patients whether they possess more than usual proclivities to rheumatism and the like, we also inquire respecting various other states. Has he been injured by malaria? Has he been exposed to the causes which produce bronchocele? Has he a tendency to cancer? Is he gouty? Has he any permanent peculiarity of the circulation giving tendency to local disease, or of his blood-vessels causing risk of hæmorrhage? In these and various other directions we see our way to the construction of a host of diatheses of less importance than those we have mentioned only because they are less common. In the present lecture I shall glance at certain general groups of diatheses, arranged according to their causes, and in the two subsequent ones I shall take various special diseases, or isolated symptoms, and examine in respect to each how far it is possible to make use of it as a clue to the diathetic state pro-

ducing it. Our knowledge of many of the diatheses is for the present far too vague to admit of our attempting any satisfactory classification of them, and a further difficulty occurs in the fact that their differences are often very dissimilar in kind. We might suggest a group of those which are transitory and those which are permanent, and others of those which may be acquired, and those which are solely obtained by transmission. It is clear, however, that any such grouping as this would often necessitate our placing the same malady in more than one class. On the whole, I think the simplest plan will be to make for the present a rough classification of the causes of diatheses, a task far easier than that of the diatheses themselves, and following this, I shall bring before you a few examples of each.

I will commence first with diatheses in relation to *climate.* Under the term *malaria* we may include all the various emanations from marshy grounds, swamps, and jungles. As to its exact nature we know nothing, but that, whilst similar in essential qualities, it varies in different localities, both in intensity and character, seems certain. Its effects upon the human constitution may be to produce quickly very severe disease, but, whether acute or otherwise, attacks of arterial spasm attended by visceral congestions are almost constant phenomena. Its effects, whether exposure be continued or otherwise, are always permanent. The man who has once had ague will display through life peculiar susceptibilities. Under all circumstances likely to cause shivering he will show especial tendency to rigor. He will shiver if a catheter be passed, or if he be exposed to cold, or if inflammation is initiated; and it becomes a matter of some practical importance to the operating surgeon to remember that rigors occurring to such patients are not so serious in their import as in others. They are retrospective symptoms, and reveal the fact that those to whom they occur have at some former period come under the influence of malaria. One attack of malarial fever by no means prevents another, and although subsequent attacks are usually less severe than the first, yet the effects of the poison are distinctly cumulative, and the limits within which acclimatization can secure immunity are probably much more narrow than has been supposed. It is easy to see that a poison which can be so persistent in its action and of which the effects, even in mild cases, are so well nigh permanent,

must be capable of producing that state of body which we name diatheses. The malarial diatheses is, indeed, a well-marked one, and it exists in greater or less degree in all who have ever come under the influence of its cause. It is probably in some degree hereditary, the inheritance being, we may suppose, in ratio with the severity and length of duration of the disease in the parent. Inasmuch as the offspring of those who have suffered are usually born under conditions of continued exposure to its cause, it is difficult in them to discriminate between what is inherited and what is acquired. So great, however, is the combined influence of the two that a fearful state of physical degradation may be thus produced, and in this the inhabitants of a whole district may be involved. Nor is the physical health alone affected; the nervous system is especially involved and the mental and moral faculties are largely influenced. When we remember how permanent in hereditary transmission such influences become, we can easily understand that the malarial diathesis has, in many instances, had a large share in controlling the destiny of a nation or damaging the character of a race. This diathesis can easily mix itself with any other, and in London practice we encounter it very frequently in association with that of syphilis, and occasionally both with it and that of alcoholism. I am speaking, of course, not of London residents, but of those who have lived abroad.

A climatic diathesis which must be placed side by side with that due to malaria is the one which results in bronchocele and cretinism. Here, again, we are ignorant of the precise cause, but we know that it appertains to certain local telluric conditions, and especially to mountains and valleys, just as we know of ague that it belongs to marshes. The cause of this diathesis is much more slow in its effects than is malaria. Far longer residence in the locality is required, and for its worst effects it appears to be essential that there shall have been transmission through several generations. It appears to persist with considerable tenacity in families which have once suffered from it. Like malaria, it degrades the entire man, and whenever prevalent produces certain deterioration of race. This diathesis may perhaps be most conveniently named, from its most conspicuous symptom, *the bronchocele diathesis.* It is absurd in the case of this endemic disease to speak of

bronchocele only, since the general health is always at the same time involved. At the risk of being accused of repetition, I must here again insist upon the features of distinction between diathesis due to food and those to be assigned to climate. The latter are distinctly restricted and endemic; the others not so. The latter, when severe, affect all immigrants who come within their range of influence, without regard to health, position, or habits, and under certain circumstances leave scarcely any exempt. The diet-diathesis, on the contrary, as illustrated by leprosy and the like, affect immigrants only very seldom, and with great apparent capriciousness, taking one and leaving a thousand untouched.

These diet-diatheses constitute a group of great importance. They are persistent constitutional conditions—sometimes hereditary, at others not so—which have their origin in connection with food. Of them by far the most important for us, as English surgeons, is gout. Perhaps I ought to have assigned the foremost place to scrofula, and I should, indeed, have done so, but that it probably acknowledges a mixed causation. In many gouty patients the sum total of the diathesis is, as regards its causes, mixed. There may be susceptibility to weather, and to influences on the nervous system, as well as to articles of food, but these we can separate from what we mean by gout itself; and so far as this is concerned I believe we must all acknowledge that it is a question of diet. About the intermediate steps of causation there may be room for much discussion and difference of opinion. But I suspect that no one will decline to accept the suggestion that had mankind continued to be vegetable-feeders, and never known the use of wine or beer, we should have had no experience of gout. If this be granted it is all I ask, for it is to be freely admitted—nay, expressly asserted—that such a thing as uncomplicated diet-gout is now scarcely to be met with. If, however, we can succeed in any degree in separating its complications from itself, we shall find in gout the best type from which to study the laws of a diet diathesis. In my own mind, leprosy stands next to gout in the certainty that it is due to food, but I cannot speak as to this theory of its origin as being yet commonly admitted.

Certain general statements may be expected to be found true concerning all diatheses which are due to food. They

ought to be the same or nearly the same, in all countries where the same diet is used, and they ought to cease where the diet is wholly changed, and to undergo modification, or to become rare, when it is modified. To some extent they ought to be curable by abstention from the articles of food supposed to be their cause; but we must not push this too far, since many constitutional conditions, when once established, may persist, or even become hereditary, after the removal of their true cause. I scarcely need recapitulate the facts respecting gout which prove its association with dietetic habits. They are too well known. It may be well, however, that we should be reminded that the diathesis, when once established, becomes by inheritance remarkably persistent, and that it is unquestionably the parent, when so transmitted, of many very peculiar maladies of which the affiliation might easily have been overlooked. I went into some detail in one of a former series of Lectures in describing a peculiar form of destructive inflammation of the eyeball, a chronic and almost painless irido-cyclitis—which occurs only in those who inherit gout, and I shall not now say more respecting this disease than that I have, since my former lecture, seen some remarkable cases which supported the views then propounded, and none at all which were exceptional to them. There are several other less definite, and still less common forms of eye disease which stand, I believe, in the same relation to gout, and with them I would ask to be permitted to place (and I speak not without the justification of carefully-collected evidence), a large number of cases of crippling rheumatism, almost all those which are known as spondylitus deformans, a great majority of the cases of gonorrhœal rheumatism, most of the severe forms of neuralgia occurring to young persons, and many peculiar liabilities to hæmorrhage. It seems, indeed, under the ordinary customs of English society, very difficult, even after several generations of strict temperance, to get rid of the gouty diathesis, and some facts would dispose us to the belief that, even after it has ceased, perhaps, during several generations, to manifest itself in the typical form of joint disease, its effects may still be encountered in predisposing to disease of blood-vessels, of the kidney, and of the nervous system.

The facts regarding leprosy are probably somewhat different, but some allowance must perhaps be made for the cir-

cumstance that we have not as yet had an opportunity of examining them so critically. It would appear to be far easier to produce leprosy *de novo* than it is to produce gout, and it appears also, as might be expected from this fact, to die out of the system and out of the race much more quickly than does gout. Probably it is associated with some much more definitely specialized dietetic poison than that which causes gout. For, if the hypothesis which I advocated before you two years ago, and in which I believe with increasing firmness, be correct, it is certain that the leprous poison is capable of considerable concentration under favorable circumstances, and may produce its effects in a comparatively short space of time. Many cases might be cited in which children or adults of healthy English families residing for a few years in a leprosy district, and eating only the ordinary food of the place, have become its subjects. Even in these, however, the malady is never rapidly developed. It always comes on slowly, and not unfrequently does not reach its height until possibly several years have elapsed after removal from the place where it was acquired and return to English food. This development of leprosy without possibility of hereditary transmission, and after short periods of residence in dangerous localities, is, I believe, never witnessed in temperate regions, and never excepting where it is well known that unwholesome varieties of fish are met with, and where also the wholesome kinds are liable to rapid decomposition. The fish theory assumes that, under such conditions, the special leprous poison may be taken in very efficient quantities in connection with a very small allowance of fish. It is the quality of the fish, and not its quantity, that we must regard. Next I stop to note that the same fact is observed, though not to the same extent, in the case of the beverages which produce gout. I am especially anxious to draw attention to this statement, because it is almost always forgotten or ignored by those writers who still disbelieve in the fish hypothesis.

As regards my suggestion that the leprous diathesis is not only more easily produced than that of gout, but dies out more quickly when the cause is removed, many interesting facts might be mentioned. Almost all authorities on the disease have laid stress upon its hereditary tendencies, but a great fallacy underlies all their observations in that these

supposed tendencies have been witnessed in districts where the original cause—supposing that cause to have been a poison taken in connection with fish—was still in existence. Thus, it is easily possible that the inherited tendency may have been, at any rate, recruited from time to time by the original poison. The careful observations of Norwegian physicians in the United States have proved that leprosy does not persist in the families of immigrants from the leprous districts of their native land, and have further, I think, made it probable—although I do not know of any special cases that have been published in proof—that even the subjects themselves of leprosy get well after a long residence in their adopted country. I had the pleasure of producing before you, at the time of the lecture to which I have referred, an old lady who had, under my own observation during a period of about twenty years, recovered from leprosy. She had contracted the disease in Barbadoes, and had recovered in England. She is still alive and in good health, and has now had no sign of leprosy for many years. This was the first case of definite recovery from a severe and characteristic form with which, at the time of my lecture, I was acquainted. Since then, however, two other examples of recovery from leprosy—or, at any rate, a cessation of all leprous processes—have come under my observation. The subject of one of these, a distinguished missionary, now an old man, is blind and crippled in all his extremities in consequence of leprosy contracted in Jamaica thirty years ago. He has lived the greater part of this time in Canada, and for many years has been quite free from leprosy manifestations. I did not myself see him when the disease was extant, but the conditions which it has left justify the diagnosis, even if it had not been placed beyond all doubt by the fact that it was made by Dr. Brown Séquard. Probably there are a great many persons living who are examples of the entire cessation of leprosy processes after long periods of residence in healthy districts; but no particular interest appears as yet to have been attracted to the search for them, and as the process of recovery is one which extends over such a long period of time it is not always easy to recover data which place the original diagnosis beyond doubt.

Before leaving the subject of leprosy I must briefly summarize the evidence anew which leads me to place it very con-

fidently among the diet-diatheses. It is the same in all countries, and probably has been so in all ages. It is the same in rich and poor, in hot climates, and in cold climates. It is certainly neither infectious nor contagious; yet it is capable of origination in those who go to reside where it is rife. It slowly gets well in those who leave its haunts and reside elsewhere. It is not possible to mention any climatic condition which is common to all the very varied localities where leprosy is found. Whenever it occurs in a temperate climate it happens to those who are engaged in the fishing trade, and who live, to a very large extent, upon the poorer kinds of fish. Although when it occurs in hot climates it usually observes the same rule, yet it now and then happens to those who eat but little fish. But this is always under circumstances where fish is known to be bad, and where it rapidly decomposes. Finally, in all temperate climates leprosy disappears before advancing civilization, the introduction of agriculture, and the increase of population.

Before we can decide as to whether *Scrofula* should rank as in the main a diathesis which is due to defective food, we have to ask what is meant by the term. In answering this question, I am glad to acknowledge help from the valuable lecture on this subject delivered before us last summer by my colleague, Mr. Travers. The general tendency of modern investigation would seem to be to associate tuberculosis with scrofula, and to teach that both may be, and usually are, the consequences of inflammatory action in predisposed individuals. Much that is known as scrofulous probably never reaches a distinctly tuberculous condition. But the name is still, I submit, appropriately applied if the inflammatory action can be shown to be of that type which often or usually results in the development of tubercle. On the other hand, there are certain cases in which the proclivity to tuberculosis is so strong that it does not wait for the advent of an inflammatory exciting cause, but is produced, as a lymphatic neoplasm, without the occurrence of preceding congestion. In, however, the very large majority of tuberculous and scrofulous cases, a predisposing cause and an exciting cause are both present; the former being an inherited proclivity, the latter an injury, a catarrh, or any influence likely to produce local congestion.

The greater the degree of hereditary proclivity the less is the need for the exciting cause. We may note also that we frequently have to witness in the case of scrofula the influence of what may be called contributory causes, such, for instance, as the occurrence of an exanthem or other acute illness. We have then to examine as to what may be the influences under which a proclivity to inflammations of the scrofulous type may arise, and we shall probably be not much mistaken if we make answer as we should do in the case of rickets, that they are largely due to deficient and unsuitable food; but that, at the same time, it must be admitted that climate, clothing, and weather take each a very considerable share. As regards diet, it is probably a deficiency of fatty matters, such as butter, oil, and the various other forms of animal and vegetable fat, which is influential; and, as regards climate, deficiency of sun and exposure to damp and cold. Scrofula, as we see it, is probably always inherited, but it is often probably largely increased by exposure to the causes hinted at. Possibly there are few practicable dietetic changes which would be more beneficial to our English population, in reference to scrofula, than a large increase in the consumption of butter and other animal fats. The comparative immunity of those who use the fish oils as articles of food, and the prophylactic virtues of cod-liver oil among ourselves, are, I believe, established facts, and their lesson is of considerable importance in reference to the dietetic causes of scrofula. We are enabled at the present time to speak much more definitely as to the diseases which should be classed as scrofulous, and as to their essential connection with the tendency to tuberculosis, than we were some years ago. A considerable number of diseases of the skin, of the bones, of the eye, have been conclusively removed from the scrofulous group, and placed under their proper designations, as cryptogamic in some cases, and syphilitic in others. So far had this gone that I can easily imagine that there may be some who think that I have given too wide a definition of scrofula, when I asserted that it is not always associated with the growth of tubercle. To such critics I will produce the instance of scrofulous ophthalmia, which usually consists of a chronic, slow-to-heal ulceration of the cornea, unquestionably dependent upon a constitutional cause, and often coincident with other scrofulous manifestations. There

are also diseases, I cannot but believe, among the inflammations of joints, which are slow, comparatively painless, in the ordinary sense of the word "scrofulous," and occurring in the relatives of those who are themselves tuberculous, but in which yet it is impossible to prove the presence of tubercle either in the bone or synovial membrane. The frequent and comparatively easy, and often permanent cure of many forms of scrofulous inflammation, is also, I think, another reason for believing that, although tubercle is its ordinary goal, it is by no means always reached. The scrofulous diathesis is indeed very ill-defined, and, in many instances, shades off very gradually into other states of defective organization. How closely, for instance, are many conditions which are called scrofulous associated with those peculiar states of feeble circulation and irritable tissues which give liability to chilblain, swollen lips, and the like affections? Yet these latter are not probably in any strong degree indicative of tendency to tuberculosis.

The liability to *Rickets*, in other words to a derangement of health, of which defective ossification is the chief but by no means the only result, should unquestionably rank as a diathesis. It is one, however, which is usually, though not quite invariably, terminable at a certain period of life. In very rare instances, so rare as to be almost doubtful, it may begin during intrauterine life, but usually its advent is delayed until near the end of lactation, and it reaches its climax, and begins to subside just at the time when the child, hitherto carefully restricted to a very few articles of food, and mainly to milk, is allowed to indulge its tastes, and to have variety. With such facts the first suspicion must certainly be that it is probably dependent on a milk regimen, and in the main no doubt this suspicion is correct. In numberless instances it has been proved that the milk upon which children have become "rickety" was distinctly poor in quality. The advantages in change of regimen are almost always well-marked, and although some experiments on the lower animals have failed in the artificial production of rickets through restricted food, others appear to have succeeded. While, however, we may safely believe that defects in diet are the main cause of rickets, and that they may be effectual in the almost entire absence of others, yet it is probable that others are usually present and contributory. In the discussion on rickets which took place at the Pathological Society two

years ago several speakers drew attention to the fact that the disease is very rare in sunny climates, although infantile inanition from poor food may be common. Mr. Spencer Watson in particular used arguments which made it very probable that free exposure to sunlight and warmth would do much to enable a child improperly fed to yet effect assimilation in such a way as to keep clear of rickets. We may certainly believe it probable that our climate and the narrow, sunless streets of our large towns take their share in the production of what was once known on the Continent as "the English disease." Let me here repudiate emphatically the suggestion which has recently come from Paris, that inherited syphilis is the real cause of rickets. The two diatheses are probably quite distinct, although they may often co-exist, and in other instances may simulate each other. Inherited syphilis,—as the labors of Taylor in New York, M. Parrot in Paris, and Drs. Barlow and Lees among ourselves, have shown us,—may frequently produce swellings about the epiphyses, and a state of general tenderness of the skull due to ostitis, which might be mistaken for rickets.

A word or two must here be said respecting a disease of the osseous system of great rarity, which has been named "Rickets of the Adult." I allude to osteo-malacia, or Mollities ossium. It is probable that maladies having relation to different causes have been confused together under this title. But respecting a certain number of the best-marked cases, there appears good reason to believe that the disease is really a form of rickets, and that the differences observed between the two maladies are chiefly due to the very different condition of the osseous system as regards its development at the time of attack. Trousseau has recorded a case in which osteo-malacia attacked a woman of seventy who had been rickety in childhood, and another case in which this disease was cured rapidly by the use of cod-liver oil and liberal diet. There is a skeleton in the Brighton Museum which was obtained through the zeal of that accomplished physician the late Dr. Ormerod, which is one of the best examples of osteo-malacia that I have ever seen. The man had had innumerable fractures, and partly through faulty union and partly through bending of his bones, had lost more than two feet of his stature. In this instance the man's children were much deformed

by infantile rickets. Although as an ordinary thing the diathesis of rickets, if not fatal, passes away as age advances, and under the influence of judicious dietetic treatment, we may reasonably suspect that there are a few cases in which it does not do so, and that it is quite possible for it to be reproduced or even initiated in adult life. In respect to its rapid subsidence, and usually its complete disappearance when the diet is improved, the rachitic diathesis has a close parallel in *Scurvy.* The latter malady is indeed of such duration that we might hesitate to apply the term diathesis to it. It is for the most part an acute diathetic disease, produced quickly and quickly cured. In saying this, however, we have not said all, for there are cases in which scurvy passes into the chronic form and becomes the source of permanent ill-health. When it does so it may fairly claim to rank with the diatheses, and it is quite possible that, in exceptional instances, some degree of influence from it may be hereditarily transmissible. In scrofula, rickets, and in scurvy, we have examples of diatheses produced by defective food, while in leprosy and gout we illustrate those due to some excess, or rather to something deleterious contained in the food. Close by the side of leprosy I ought to discuss ergotism, pellagra, chronic lead-poisoning, and some others, but time does not permit. Can the specific animal poisons become the producers of permanent diatheses? This question must, I think, be answered in the main in the negative. Unquestionably they may produce conditions of ill-health which persist for a certain time, and may cause various morbid manifestations; but these states are short-lived, and perhaps are more fitly ranked as dyscrasiæ than diatheses. This is true even, I think, of syphilis, the most chronic and most persisting of all specific fevers. Excepting in some very small degree of reduced liability to the influence of the same poison which is to be observed in the offspring of those who have suffered, I doubt whether in any case the influence of a specific fever in a parent is to be traced in the offspring. No state of health which is heritable is ever produced. Some few exceptions to this should perhaps be allowed when smallpox, measles, and the like appear to intensify the diathesis of scrofula, but they are probably of but little importance. The general law certainly is, that a specific animal poison may breed in the blood, and may for the time produce

most violent disturbance of the health of the individual, but its effects cease after a while, and cease completely, and leave the body unmodified with the exception that it is no longer liable to the influence of the same poison. This immunity lasts only during the lifetime of the individual, and cannot be transmitted excepting in an exceedingly slight degree. Even in the individual it is by no means complete or invariable. I am aware that there is an apparent exception to all this in the case of syphilis. I will speak of that directly, and will also say a few words as to the limitation of the law of immunity of the individual. Before we pass to those topics, however, it may be well to ask attention to the strongly-marked differences which are to be observed between the states of health produced by the specific poisons, and those which are due to inflammation from non-specific causes and which come under the law of intensification by habit. In the development of such inflammations as those of erysipelas, diphtheria, elephantiasis, and especially when not due to contagion, the constitution of the individual takes a large share, and the mere fact of their occurrence reveals his previous proclivity. The disease originates, in part from without, but in part also from within. It is not an instance of intrusion, but of development, and peculiarities of blood, tissue, or nervous system, are exactly those which are capable of increase. In the case of the exanthems, however, the body of the sufferer is in the main passive, and is acted upon by a poison which is intruded from without, and to which *nolens volens* his tissues must submit. The occurrence of the exanthem reveals nothing and intensifies nothing. It is from first to last a violence done from without, by an external agent, and when its direct effects are over, the body may be supposed to return in all other respects to its original state. A man who has been put in the stocks is not especially likely to acquire the habit of going to sit there, nor does the man who has been burned, or who has broken his leg, acquire therefrom any tissue-proclivities. His tissues were in no sense consenting parties, and the laws of habit do not come into play. The possibility of second attacks, and the absence of protection to offspring, are facts which indicate that the influence of exanthems is only transitory. As regards the immunity of the individual, conferred by an attack of an exanthem, we may assume that it is neither com-

plete nor universal. Modified attacks may occur repeatedly in the same person, nor are second and fully-developed ones unknown. Thus it is, I believe, a matter of general creed in the profession that, when scarlet fever is in a house, those who have gone through it before are liable to suffer from sore throat without the rash: and that with measles catarrh may in the same way denote a minor degree of protection. The modification of variola by vaccination, and also the limited duration of immunity, are well-known facts. Second attacks even of variola itself may occur.

Remembering the length of time during which the effects of the syphilitic fever may persist, we might perhaps expect that it would prove more constantly protective than the others. But the facts teach us that we must not conjecture, but be content to observe. No one has, I believe, ever categorically denied the possibility of second attacks of syphilis, but the opinion which almost universally prevailed, a few years ago, came very near to this conclusion. That, however, second infections with modified results are tolerably common, and second attacks with full development of all the phenomena, not extremely rare, is, I feel sure, the truth. I have recently had under my care, at one time, three patients in whom it was certain that they were passing through second attacks. In more than one instance the patient has been under my own observation during both attacks. One of those which I have just referred to is a very remarkable case, on account of severity of both attacks. The patient, now a surgeon, of about forty-five years of age, had syphilis when a student. It was attended by a free rash, and was followed by various sequelæ, which he did not get rid of for three years. Then he married, and he now has healthy children. In the beginning of the present year he obtained by some accidental inoculation a chancre on the lip. It became very large, much indurated, and was attended by large glandular swellings. Subsequently a most copious eruption followed, and both in primary and secondary phenomena, the case was as severe a one as I have seen for a long time. It may be that idiosyncrasy has something to do with this exceptional recurrence of liability during the individual's lifetime. In one instance,—in which I attended a member of our own profession twice for syphilis, first when a student, and secondly, ten years later,—the same person

had, he assured me, twice had variola. In respect to vaccination, it is, I believe, well recognized that some persons lose its protective power much sooner than others. Those who, by an attack of an exanthem remain protected through life, may be said in some sort to have acquired a diathesis. It is one, however, which, as I have already asserted, does not manifest itself by any active phenomena, nor is it one the influence of which extends to offspring; or if it does so, it is to an extent so small as to be almost inappreciable. It is a remarkable but undoubted fact, that *quoad* the specific fevers the offspring of protected parents starts afresh. It may be, and probably is, the fact that in successive generations the tissues become to some extent habituated, and suffer less severely, but still they do suffer, and nothing approaching to immunity is ever observed.

I have been speaking of the non-inheritance of the negative diathesis induced by an attack of any one of the specific fevers, but it remains yet to say a few words as to my assertion that none of them produce an active diathesis which is heritable. The only exception to this which will be alleged is syphilis. I shall be asked "Is not the diathesis of syphilis often transmitted from parent to offspring?" Probably it is not the diathesis which results from the disease, but the germs of the disease itself—the particulate elements of the virus—which are transmitted. It is transference, a form of contagion rather than hereditary transmission, which occurs. Two years ago I produced before you, at some length, the facts which point to this conclusion. Since then, by further thought on the subject and the accumulation of more evidence, my opinions on this subject have been much strengthened. We well know that the danger of transmission to children has reference not to the state of health of the parents, but to the lapse of time which has occurred since the primary disease. We see cases in plenty in which parents whose children have been quite healthy, subsequently afford conclusive proof that the diathesis still persists in themselves. Indeed, it is almost an acknowledged law that parents in the late tertiary stages do not transmit taint. On the contrary, when offspring are born in the secondary stage of the disease, especially in its early periods, it is a rule, with exceedingly few exceptions, that the disease is transferred. These facts strongly favor the belief,

that the inheritance of syphilis, so-called, is possible only while the fluids of the parent contain the living elements of the virus. These elements, in a large majority of cases, develop and go through the same stages as if inoculation had occurred at the time of birth. It is far more like contamination *in utero* than true inheritance. By contamination in this use of the word I mean that with sperm or germ (of either or of both parents) there passes the virus itself, the sperm or germ being itself unmodified, but simply the material medium of transference. In the case of the mother we know well that it is not necessary that the germ should have been infected at all, but that if her blood receive the virus, even so late as the eighth month, it will pass into that of the child also. Inherited syphilis, when produced under these last-mentioned circumstances, runs exactly the same course as when derived from a parental taint which existed before conception. A child then, I assert, inherits syphilis in precisely the same sense, and in precisely the same manner, as it may inherit smallpox. It inherits not the diathesis, but the disease. The reason why the inheritance of smallpox is very rare, while that of syphilis is unfortunately common, is simply that the period during which the virus is extant in the blood is very different in the two cases. The conspicuous facts, then, with which we are all so familiar in reference to the syphilis of infants, afford no proof that the diathesis of syphilis, any more than those of the other exanthemata, is capable of transmission. It remains, however, for us to inquire whether there are other facts, of a somewhat different kind, which support the view which I have endeavored to combat. I am alluding, of course, to the old controversy as to whether some forms of struma, and especially such diseases as lupus, may not be modified forms of inherited syphilis. My own belief is very strong indeed to the effect that they are not, but the facts are very detailed and very difficult to marshal concisely. My argument, if I have made it plain, has pointed to the conclusion that no minified transmission of syphilis is possible, that the child gets either nothing at all or the germs of the disease, and that in the latter case they will, subject to the laws of idiosyncrasy, develop equally in all cases.

Let me try briefly to state some facts on this question, and I will then leave it to your judgment. Nothing is more com-

mon than for parents who have themselves at some former time suffered from syphilis, to produce children who remain through life robust and in perfect health. Sometimes one or more of the family inherit syphilis, and suffer, it may be, severely, while others, usually younger ones, escape, and when this occurs the latter may enjoy almost exceptional vigor. When I have seen a whole family of grown-up children, the offspring of syphilitic parents, manifesting signs of cachexia, there has usually been reason to think either that all had actually suffered from the full disease, or that the symptoms present in some were not specific, but due to other causes. I have seen very numerous cases of lupus on the one hand, and of inherited syphilis in young persons on the other, but I have never found the two things together, nor have I ever recognized in the subject of true lupus the brother or sister of one whom I knew to have suffered from inherited syphilis. Of the cases of lupus which I have seen, in a very great number there were strong reasons for denying the probability of syphilis in the parents. I have, of course, occasionally seen inherited syphilis and scrofula together in the same patient, but a large majority of those who suffer from the former never show any tendency whatever to the latter. In what I have said above I have been speaking of course of common lupus, and have wholly excluded that form of destructive ulceration which is sometimes mistaken for lupus, but which is from beginning to end syphilitic, and is curable by specific treatment. I have taken lupus as being a type form of disease, but what I have attested, as regards it, applies equally to other forms of scrofula, to psoriasis, and other diseases, which have been suspected as the remote consequences of specific taint. I believe that the domain of inherited syphilis is just as definite and just as restricted as that of the acquired disease, and that it stands sponsor to none but its own progeny.

LECTURE V.

MR. PRESIDENT AND GENTLEMEN,—In reference to malignant new growths it is possible to use the word *diathesis* as applicable to two different things. We may mean a state of tissue-health giving proclivity to cancer, or the state which results from the existence of cancer. I will ask permission to employ the word cancer in its old and popular sense, as including malignant new growths of all kinds. This is the sense in which it has been used for centuries, and it is, I think, a pity that it should be changed, especially as we have in carcinoma, as opposed to sarcoma, a very convenient term by which to distinguish for scientific purposes certain forms of malignant new growths from others. In examining as to diathesis, we must merge all the distinctions which have been established by the splendid labors of modern histologists, and speak of malignant new growths as one family. Indeed, it is by no means improbable that some forms of new growth not counted as malignant, ought in this matter to be placed with those which are so, and that we should estimate the individual tendency to new growth in general without paying too much regard to differences of kind. We not infrequently observe that some persons display more than one form, and when hereditary transmission occurs it is very common to see it produce a growth of a kind different from the original. On this doctrine of transmutation in transmission I had much to say in my last year's lectures.

The term diathesis is, I think, to be repudiated, as not suitable in reference to liability to the general diffusion, local recurrence, and failure of health which the existence of cancerous growth produces. These are all the direct and immediate effects of the disease, part of it, and are better named the cancerous dyscrasia. The real question before us is whether there exists any peculiar state of health wholly antecedent to the development of any local growth which

gives proclivity to it. If such exists, and especially if it be in any way recognizable, the term diathesis may be conveniently applied to it. The first observation to be made in reply to this question is that cancer in the main is obviously dependent upon age, that it is in nine cases out of ten part of senility—a sort of second childhood of the tissues. Among the lower animals yet more than with ourselves, cancers are, I believe, almost always the appanage of age. Old dogs and old cats get cancer of the lip or mamma, but these maladies are scarcely ever seen in young animals. When new growths which approach the malignant type are met with in the young there is probably almost always a history of inheritance. If we admit this statement we admit, at the same time, that there is a cancerous diathesis; since it is clear that a state of health may be transmitted which gives proclivity to the disease, without actual conveyance of the cell germs of the new growth. The probability that such states of tissue-health exist, is increased when we take cognizance of the numerous cases in which, with strong hereditary history, no manifestation occurs until adult or even senile periods of life are reached, though it must be admitted that these facts may be held to be evidence in both directions. We shall probably be not far from the truth if we admit senility of tissues, local or general, to be the one predisposing cause of cancer with which we are acquainted; while injuries and all forms of local irritation are its exciting causes. A little step further may next be taken, in the belief that everything which tends to hasten senility, either local or general, will increase the predisposing influence, and in this category may be placed anxiety, distress, over-work, and excesses of all kinds. It is highly probable that under such conditions a state involving increased proneness to cancer may be induced, and further, that if offspring are produced after that state has been developed, they will inherit that tendency. This leads me to say a few words as to the last-suggested cause of diathesis which I have placed in my list. It may have seemed strange to some that it should have been even hinted that senile changes can ever produce anything worthy of that name. It was by no means my intention to propose to re-christen old age by the name diathesis. What had occurred to me was rather this: Are there not conditions of tendency to premature old age which amount

almost to disease and which are sometimes observed to run in families? The results are simply general tissue-degenerations—atrophies, arcus senilis, white hair, pale blood, and general failure of nutrition—and yet the causes are to be sought among the ordinary causes of disease. Both arcus senilis and white hair, when met with, as they sometimes are, early in life in many members of the same family, and wholly without other accompanying signs of senility, may be held to rank as idiosyncrasies of tissue, and imply little or nothing as regards the future. But there are many cases in which they are attended by other conditions, and in which they imply much. It is possible that arcus senilis may be in the course of hereditary transmission sometimes wholly detached from the state of health which produced it, and may become, as we have suggested that hæmophilia and xanthelasma sometimes are, heritable *per se*. More usually, however, it is probable that other conditions, such as tendency to arterial disease, accompany it, and that the total result is a state of premature senility. Certainly there are families in which a tendency runs to early senility rather than to any specific disease. I recently had occasion to amputate the lower extremity for senile gangrene of the foot of a gentleman who was barely fifty, but in whom general calcification of the arteries had proceeded to an extreme degree. I cannot but think that it would be convenient to recognize as a diathesis the state in which premature senility amounts to a disease.

I wish next to ask attention to certain laws as to persistence, self-aggravation, and recurrence, under which morbid processes, due in the first instance to simple causes or possibly quite accidental, may in the end breed diathesis. The best examples of this, which I shall be able to find, will perhaps be in such maladies as catarrh, erysipelas, and elephantiasis. Indeed, in what I said as to carbuncle, diphtheria, and erysipelas, I have already glanced at it. In our last lecture I contended that the specific fevers, in virtue of their own specificity,—and that they are intrusions from without rather than modifications of existing elements and processes,—possess no power of originating pathological habit, and thus do not become the parents of diatheses which are permanent. After a longer or shorter time their influence ends. In the instance of syphilis the length of reign is undoubtedly pro-

longed; still it does eventually terminate, and terminate completely. In my argument on this point I suggested that there was an essential difference in respect to this between inflammations which begin from particulate contagia and those set up by other influences, inasmuch as the latter do tend by the mere fact of existence to develop what we call pathological habit, and to increase in power by the mere fact of possession. It is, of course, not meant that they always do so, for fortunately there are other vital forces which are constantly working in other directions and tending to the restoration of health. What I wish to hint is that there is always a risk that an inflammation once experienced may become more or less habitual, and that its tendency is, if not counteracted, in that direction. In some minor degree this is true probably of all inflammations, even of those which have been caused by injuries. There are several ways possible in which local inflammations or local injuries may become the causes of permanent change in the general health. They may in the first place, by the long-continued pain and irritation caused, reduce the nerve tone, and thus stamp a lasting impression on the cerebro-spinal part of the organization; or secondly, by long persistence they may influence alike the blood and the nervous system. Sometimes it is not persistence so much as tendency to frequent recurrence which effects a similar mischief. Lastly, I shall have to maintain a theory that all inflammatory action is in itself infective, not to the same extent, but after the same pattern as malignant new growths. On this latter point permit me to go into a little detail before I proceed to the others. It is, I am sure, a fact familiar to all surgeons that, when acute periostitis attacks one bone, other bones are very prone to follow. Many years ago this curious fact attracted the attention of Mr. Simon, who wrote on it under the name of "necrosial fever." The affection of the other bones is distinctly secondary to that of the one injured, and not simultaneous. There is usually a considerable interval; and further, those bones which suffer latest never suffer so severely as the first. It is exactly as if from the bone first inflamed some elements had been shed into the blood having the power to infect others. The bones affected secondarily may be in no structural connection with the one first attacked—may, indeed, be at a great distance from it. Thus, I have seen it begin in the tibia and

occur secondarily in the humerus, scapula, and ulna. The risk of implication of other bones exists only during the early stages of the disease, and is, I think, very seldom witnessed after the first fortnight. Mr. Simon's term, "necrosial fever," might perhaps suggest similarity with the form of multiple acute arthritis which we name *rheumatic fever.* In the latter, however, many joints are affected simultaneously and symmetrically, and all suffer in equal severity. In these features rheumatic fever differs much from what I have been just describing, and suggests that the different joints are affected independently of each other and from a cause common to them all. It is quite possible, however, that even in rheumatic fever the law to which I have adverted may be in force. We may remember that sometimes the arthritis does for a short time restrict itself to a few joints, or even to one, and that often some joints which escape at first suffer later on, while the implication of the heart never happens until a considerable time after the first outbreak in the joints. These facts would fit well with the theory that the parts first inflamed contaminate the blood and become the causes of inflammation of similar tissues elsewhere. It may be that it differs chiefly from necrosial fever in that the process is much more rapidly accomplished. At any rate as regards multiple periostitis we may be sure that in it the multiplicity is not to be explained by supposing that there is from the beginning some common cause, since in nine cases out of ten the inflammation of the bone first affected was caused by an injury. All who have paid attention to the development of eczema cases will, I am sure, bear out what I am now going to assert. It is very common for eczema to begin locally, from a known local cause, such as the use of an irritating lotion or poultice, the employment of washing powders, or exposure to heat, and for the eruption subsequently to become general. It is possible that the restriction to the part first affected may be very short, or, on the other hand, it may be very long. I feel that I may almost venture to extend the scope of my first assertions and to say that I believe that it is rare for a local eczema, if allowed to persist, not to become sooner or later a general disease. It would be easy to collect instances innumerable in which eczema beginning around an ulcer on one leg has been followed by eczema on the other quite independently of any

ulceration. It is not extension by continuity of tissue or even by contiguity, but reproduction of the same type of inflammatory action in the same tissue at a distant part. In almost all cases of general eczema there is the history of an early period in which it was restricted to one part. I have several times seen diseases of the finger-nails (chronic onychitis) appear on several fingers and on both hands, apparently in consequence of an injury to one. In these instances there is always, I think, a long interval between the injury and the affection of the other fingers, and during the interval the injured nail has always remained in a state of chronic inflammation. A gentleman who had consulted me a few weeks ago for thickened and opaque nails, and in whom several on both hands were in various degrees affected, told me respecting one which in no respect differed from the others: "Oh, that has been so for thirty years; it was from a crush." I found that the crushed finger nail had been diseased for twenty years before the others began to suffer. He had no skin disease, nor was there any history of such in his family, but I must add that he believed that his grandfather had had some affection of the finger nails. It will be thought by many that this latter fact deprives the case of its supposed significance and reduces it to simply an example of hereditary tendency. I have no doubt that in most cases in which local inflammation shows a power of contaminating the system we must acknowledge the existence of a predisposing cause as well as the exciting one, and it is to this *rôle* chiefly that I should incline to assign inheritance in the case just narrated. No doubt the cases in which local eczema becomes general happen in persons who have a proclivity to eczema, but this fact only reduces and by no means takes away the importance of the exciting cause, since without it, in all probability, the morbid phenomena would never have come into existence. The occurrence of eczema in any part and in connection with any local cause, must rank as a revealing symptom to this extent, that it makes known the fact that its subject is one liable to eczema; but itself must next, if what I have suggested be correct, rank as a cause, and a very efficient cause, of aggravation of the previously existing proclivities. Under this suggested law of self-aggravation the permitted persistence of any local disease may become a source of danger, just as

we well know the permitted persistence of a malignant growth constantly does. Nor surely is there any *à priori* reason why the life tendencies of inflammatory products and of those of new growths should be wholly dissimilar in this respect. Among other instances of maladies which often begin singly and locally and subsequently become multiple or general, we have boils, sycosis, common lupus, various scrofulous affections, and certain forms of rheumatism. Respecting all these I believe that it is true that if they are cured after having persisted a long time, the patient remains far more liable than others to a fresh development. In some sort a diathesis has been produced. In the case of periostitis I have seen some remarkable examples of this.

We have now to consider the laws just adverted to, in relation to those which regulate the recurrence of inflammatory attacks. That certain forms of erysipelas are prone to become persistently recurrent is, as I have already observed, well known. We habitually recognize the fact that some patients are "liable to erysipelas," and expect that in them attacks will be produced by slight causes and that they will prove of much less severity than first ones usually do. If the recurrence has been very frequent the attacks are usually very short and mild; sometimes the intervals are so short that the disease becomes almost persistent. Exactly the same facts are observed as regards proclivity to catarrh in all its varieties. They are sometimes also seen in liability to repeated attacks of diphtheria, a fact which has been recognized by many writers. I had recently occasion to perform tracheotomy in an orphan school on a child who was *in extremis* from croup. In conclusive proof of the nature of the disease we removed at the time a large fibrinous cast of the trachea. The child had passed through, I was informed, four attacks of croup previously. It recovered after the operation, and has, I believe, since been threatened with a sixth attack, which, however, never became severe. There is a peculiar form of œdema of the eyelids and cheeks which is caused by repeated attacks of erysipelatous inflammation, and which results at last in permanent deformity. Of this I have seen many examples. In these cases the first attack is usually one of the ordinary form of erysipelas of the face, with the liability to indefinite extension constantly witnessed in the malady; but the

later ones are limited, both in duration and extent, and the patient at length comes to know what to expect, and to be confident that the swelling will not go further than the face, nor last longer than a week or ten days. In some persons, and especially, I think, in the young, the upper lip and the alæ nasi are affected rather than the eyelids. The type of inflammatory action sometimes, as may be inferred from what I have said, ceases to be the most characteristic of erysipelas, and in others from the beginning it has in minor features departed from that type. Thus there is a form which we might call vesicating erythema which often affects the cheeks symmetrically, is of limited duration, and occurs over and over again. In some instances the vesication is extremely slight, or possibly absent, and erythema with œdema is all that we see. The cases differ from erysipelas in that there is little or no tendency to spread at the edge, and in some instances, in the comparative absence of œdema. They occur I think, chiefly to the young, but adults and elderly persons are liable to recurrent inflammatory attacks of parallel clinical history in which the type of disease is rather that of an acute but transitory eczema. All those diseases appear to be for the most part catarrhal in origin, that is, the exciting cause is usually exposure to wind or cold. They are of great interest to us as proof that maladies which are probably due to very similar causes both predisposing and exciting may yet be attended by differing outward manifestations, and that, when once the type has been taken in the individual, the recurrences will be all of the same character. They also form interesting pathological links one with another, and between erysipelas and its allies. The morbid conditions which I have been referring to certainly establish in the individual who is their subject, through their long persistence or frequent recurrence, a state of health well deserving the name of diathesis, and there can be little doubt that the peculiarity so produced might, and would be likely to, become hereditary. We thus have occasion to note how a permanent and transmissible peculiarity of health may take its origin. Erysipelas has in elephantiasis a congener of great interest. The distinction between the solid œdema which results from recurrent attacks of erysipelas and the more characteristic form of elephantiasis is only a matter of degree, and it is well known as regards

all elephantiasis that repeated attacks of rigors with erysipelatous swelling are constant features of the disease. One is tempted, indeed, to go the whole length, and declare that elephantiasis is, after all, only an example of persisting, or exceedingly chronic erysipelatous inflammation with its resulting hypertrophic changes. Elephantiasis may vary infinitely in degree, but not at all, so far as I know it, in kind. It always begins in œdema, and from this it slowly progresses to hypertrophy. The hypertrophy may implicate the corium and cellular tissue only ("smooth elephantiasis"), or it may involve also the papillæ, and produce the tubercular form. Usually these two forms occur together in different parts of the same limb. The solid œdema, which is its first stage, may begin from a variety of causes. It may be induced by an attack of *bonâ fide* erysipelas, by an injury, by any slight local inflammation, a chancre, an excoriation between the toes, etc., etc. Almost invariably its persistence is favored by the mechanical disabilities of the part, as regards the return of the venous blood and the contents of the lymphatics. We meet with elephantiasis chiefly in the legs, the labia, clitoris, penis, and scrotum, parts which, if once they become swollen, are dependent. The non-symmetry which prevails in a large majority of cases denotes the influence which purely local inflammation has in locating the disease. In some cases it affects equally both legs, and in these it may be taken for granted that the constitutional predisposing cause is strong. Such cases prove the fact of hereditary diathesis. They are scarcely ever met with in England, but only in those climates where the disease is endemic, and in races or tribes which are peculiarly liable to it. When the elephantoid process has once well set in I believe it is never wholly cured; and no better instance could be produced of the pathological advantages of possession. The worse the disease is, the worse it is likely to become. It is emphatically a self-aggravating malady. The mere fact of its existence tends necessarily to its spread. Slowly but surely it undermines the health of its subject, impoverishing his blood, and mainly by the recurring attacks of erysipelas which attend it, enfeebling his tone. Constitutional treatment does but little, and local measures are the only chance of benefit.

We need not feel much difficulty in interpreting the phe-

nomena which we witness both in recurring erysipelas and persistent elephantiasis. They are doubtless examples of the pathological power of habit and indulgence. Just as a man who has yielded to intemperance is in danger of becoming a drunkard, so it is with the tissues. The oftener they have yielded to any special process of inflammation, the more prone are they to yield again. No doubt but that, as in the case of the drunkard and the epileptic, so also here, pre-existing susceptibility takes its share in favoring both the first outbreak and the continuance. This share, however, probably varies much in different cases and may in some have been but very small. The main power at work has doubtless been the increase of proclivity which every recurring attack begets, until at length the habit or the disease, call it which we will, becomes fixed. Nor must we consider, as I have already remarked, that this power of habit will be restricted in its influence to the individual. If children be borne to a man after the habit has been acquired, they will almost necessarily inherit it. The certainty of inheritance will be in ratio with the fixity of the habit and the length of time that it has persisted before the birth of offspring. A great difference must be noted as to the inheritance of proclivities which are developed before and after the conception of offspring. If a disease comes into active existence only subsequently to the production of the whole of the offspring of its subject, clearly the latter will inherit only the degree of proclivity—and it may chance to have been but very small,—which their parent may have originally possessed; but if born after the diseased process has been long in activity, they will take over the original disease much augmented under the force of pathological habit. Illustrations of this in the case of the children of drunkards are of common occurrence. It is all the difference that there is between one who simply bequeaths his patrimony as he received it, and one who has largely increased it by his own gains. Elephantiasis, although precisely the same disease, whether met with in temperate climates or in the tropics, is far more common and severe in dark races and in hot countries. Malaria has, perhaps, something to do in predisposing to it, and heat and moisture unquestionably aggravate it. It is possible, however, that we have much exaggerated our impressions both as to race and climate as strong influences, since

the laws of inheritance will account for excessive prevalence to a large extent when once it has obtained its hold. It may be that we should regard it as to a considerable extent resulting from a diathesis which is heritable in certain families, tribes, and races.

There are certain phenomena which may, perhaps, deserve the name of *Revealing Symptoms.* Not but that all symptoms are in a sense revealing, but these in a special and emphatic manner, since they disclose the fact of the existence of a tendency, which, although present from birth, possibly has never been suspected. Symptoms of this kind occur, as might be expected, in connection with powerful exciting causes. Peculiar manifestations following the employment of special drugs, or the introduction of special animal poisons, may rank under this head. If a patient who receives the virus of small-pox developes a hæmorrhagic eruption, or if acne follows small doses of iodide of potassium, we may feel sure that in these occurrences we have a revelation as to the peculiar idiosyncrasies of the individual. It is certain that those to whom these events happen differ and have probably differed through the whole of life, from others in whom similar causes are not followed by similar effects. The touchstone by which these individual peculiarities may chance to be revealed are innumerable. The behavior of the vaccination sore and that of the primary sore often furnish to the observer an unexpected clue to the peculiarities of the individual. Sometimes the revelation concerns only the specific cause which has been in action, but in others it extends much more widely. For instance, I believe that a clean, well-indurated chancre without tendency to ulcerate or inflame rarely occurs, excepting to a person in sound health, and is far more common in those of dark complexion than in the fair. An opposite tendency on the part of the sore by no means always reveals or implies weak tissues or poor health, but it often does so in a definite manner. In making inferences of this kind, however, we must guard against the error of attaching importance to the state of the sore during the first week, which is usually due rather to peculiarities in the pus by which the contagion was effected, than to those of the recipient. The syphilitic virus itself is, on the other hand, always probably the same, and differences in its results are due to temperament, diathesis, or health-

state of its recipient, and come into the class of revealing symptoms.

Some of these phenomena, deserve, I think, another distinctive name, and may be styled as *Retrospective Symptoms.* They reveal not that which is to come, nor even that which is present, but rather that which is past. In some cases it may be that their causes are still extant and influential, but in many I believe that the symptoms in question occur only at a considerable period after the termination of the activity of the influence which gave it birth, and that they imply little or nothing as to the present health of the patient or his future liabilities. They may, however, reveal a diathetic tendency, which by hereditary transmission may bud out anew in offspring. The explanation of this probably is that they are associated with diatheses which manifest their existence chiefly in middle or early periods of life. The term, *retrospective symptoms,* ought not, perhaps, to be applied to conditions which are simply the remains of bygone disease, however useful in a like way such conditions may be. It is rather to be kept for conditions which originate late, and which, like slow-growing seeds, spring up long after the seed-time. The best example which I can give of what I mean occurs in the instance of xanthelasma palpebrarum. When xanthelasma is developed in various parts of the body in an adult, I believe it is always associated with liver disease, and often with definite and severe jaundice. In these instances it reveals a still extant hepatic diathesis, plus probably some slight tendency to new growth and fatty degeneration. In the far commoner cases, however, in which xanthelasma patches are met with on the eyelids only, no such association is observed; a large majority of those who show them have never had jaundice and are in no sort of danger of it. Many, however, have in former years suffered much from bilious headaches, and if not actually from these, they have experienced repeated temporary health disturbances, attended by dark areolæ around the eyes. It is this fact which the patches in question reveal, and they may occur for the first time long after all liability to the disturbances in question have ceased. They are, therefore, retrospective, and I am inclined to think that in many cases they are solely so, and that little or nothing is to be inferred from them as to the patient's future. In other cases—and perhaps

not a very small minority—the original liability still persists, and will continue to show its effects. But even in these, xanthelasma patches are usually the product of what is long past, and have required a very long time for their production. I have already spoken of certain other and very rare cases in which xanthelasma occurs in young children—possibly in several members of the same family—always in a multiplex form, and without any association with disturbed health. In these curious cases I would suggest that the retrospection is yet further back, and that they reveal the fact that some predecessor has suffered from liver disorder, or possibly been the subject of xanthelasma itself.

It is true of a number of morbid states which have received special names, and are often spoken of as if they were complete in themselves and distinct from all others, that their chief interest for the nosologist lies in the fact that they are symptomatic of diathesis. Their occurrence reveals the states of peculiar health into which the persons showing them have respectively fallen. Thus, as we remarked when dealing with the subject, enlargement of the thyroid gland, when endemic, reveals a diathesis which may end in great degradation of the physical state generally, and we ought by no means to be content to name it bronchocele, as if in that term we had denoted the whole malady. I propose now to take up in succession a number of names which denote according to custom distinct diseases, and to ask concerning each what is its symptomatic value, what does its occurrence reveal, how far may its existence help us to the discovery or recognition of the health peculiarities of the individual.

We will take first *Acne*. Under this name conditions which differ much from each other are grouped, but my impression is that the grouping is to a large extent natural, since they really are closely related. All forms of acne have this in common, and they are due to morbid processes occurring in or around sebaceous glands, and further, that their commonest site is the face, with the exception, perhaps, of white acne or milium, which is due to congenital occlusion of some of the gland orifices and accumulation of white sebum under a thin transparent pellicle of epidermis. All are, at one stage or another, attended by congestion or inflammation. Their differences depend probably upon differences in the character of the

skin in different persons, differences in age, and modification in the ordinary causes. In order that acne should occur, it is essential in the first place that the sebaceous system should be largely developed, and the skin moderately thick. These conditions given, we shall then be able to watch the play of various influences, local and general, upon their subject's health in producing its various forms. If his state of tone and vigor remain perfect, probably his glands will continue to elaborate and pour forth their secretion, without any disturbance. No accumulations will occur, and no secondary congestion around the glands will be observed. But the slightest, the most temporary disturbance of tone may derange the function of these glands, and may permit the retention of a thick secretion, or favor the occurrence of inflammation around an irritating plug. How rarely do we witness the occurrence of acne in any form before puberty, and how frequently do we obtain proof, after that period, that the influence of the sexual system is all-potent in so disturbing the tone that acne spots are produced. In girls menstruation is often attended regularly every month by fresh eruptions of acne, and in boys nocturnal emissions frequently have the same effect. If, however, the integument were originally thin and not greasy, then it is possible for the tone to be very seriously damaged by the causes adverted to, and yet no acne may be produced. In such cases pallor of skin may be the only result. In others in which there is less than usual proneness to inflammation, and perhaps very slight disturbance of tone, a peculiar form of lichen-acne, or very chronic persisting enlargement of the glands without congestion, may occur. This is seen chiefly in the temples and forehead, and in the male sex it is often coincident with similar long persisting enlargement of the glands on the penis and scrotum. The location of acne on the face is probably often explained by pre-existing peculiarities in the state of the skin of the face. Some get acne almost solely on the chin, or on the chin and cheeks, and these are almost always those in whom the nose is specially thin. On the other hand, those in whom the nose and skin generally are thick become liable, when acne is developed, to have the cellular tissue around the glands implicated as well as the glands themselves. Thus, tuberose indurations of a very chronic nature may be produced, which, if in adult life the causes of

aggravation remain, may advance from what is known as acne tuberosa to the most grotesque deformities. Acne is very constantly hereditary, the same form often prevailing in several members of a family, and acne tuberosa, I believe, often descends in several generations from father to son. I have not seen acne tuberosa more than once in women, and in that instance a sister of the patient had common acne, and her male relatives had shown the tuberose form. The pustular or common form belongs to youth, the rosaceous or erythematous to adult life. If, then, we attempt to reply to the question, "What does acne in its various forms imply?" we should, I think, have to answer that in the first place, it denotes original and heritable peculiarity in the structure of the skin; next, that its common form in young persons usually implies greater or less disturbance of tone in connection with the sexual system, and that its rosaceous form results from dyspepsia, attended by flushing of the face after meals. The tuberose variety implies original peculiarity of structure, and is often aggravated by dyspepsia and intemperance. Common acne is almost constantly attended by proofs of enfeebled circulation, such as cold feet, and often by constipation. Closely associated with acne is the liability to styes, to some forms of sycosis, and to boils. With these exceptions I know of no forms of skin disease which are due to the same class of causes or denote the same conditions of health.

A few words may next be said as to the symptomatic meaning of *Eczema.* This symptom is in a large majority of instances so far local that it is curable by local measures, and scarcely, if at all, by constitutional ones, whether drugs or restrictions as to food. Yet it is probable that there is always a minor degree of constitutional proclivity and this is sometimes proved to be hereditary. In a few cases, dietetic restrictions do appear to have important influence, as for instance, the forbidding of milk and sugar. I have already alluded to the remarkable way in which eczema appears to aggravate itself, and when once it has begun is its own source of extension. Probably a great many cases which become severe and general, might have been stopped in the beginning by appropriate local treatment. In most forms of eczema arsenic is useless, and this fact serves to detach it definitely from the psoriasis group. There are, however, certain forms

of nummular eczema in which well-margined patches are scattered symmetrically over the limbs and trunk, in which the disease approaches very closely to a form of psoriasis, and is more or less under the control of the specific for that disease. Putting aside a very large number of mild or local cases, which are clearly due to local causes, we encounter severe eczema in the following forms: First, as a disease of the dentition period of infancy, or what is often equivalent, the lactation or milk-food period. Second, as a most persisting and troublesome eruption affecting only special regions in children and adults, as for instance, the hands, the lips, and the anus. Thirdly, as a general and severe eruption in advanced adult or senile periods of life. It is a noteworthy fact that when infants who have suffered very severely get well, they usually get quite well, and remain well through life. General attacks affecting the whole body occur for the most part near the extremes of life. Applications containing tar, if weak enough, will almost always both prevent and cure eczema. Sea air is often definitely advantageous, and the disuse of milk and sugar are often important. With such facts before us can we find answers to the questions:—Is eczema usually a sign of gout, or of any allied condition of defective digestion? Is it catarrhal? Is it due to structural idiosyncrasy of the integument? I should incline to reply that it is certainly not catarrhal in any correct use of the word. It is not produced by the common causes of catarrh, nor does it display the clinical course of all catarrhs, in the tendency to spontaneous recovery and frequent repetition. Next in many cases it does imply a minor degree of mal-assimilation allied to gout, and is benefitted by abstinence from beer and wine. Recent experience has led me to believe that the offending article is often milk, and to think it of importance to restrict it as much as possible. In very many, a large majority of cases, there is no true gout either in the patient or relatives.

LECTURE VI.

MR. PRESIDENT AND GENTLEMEN,—There is yet one other skin disease of such extreme interest in varied directions in reference to questions of diathesis, that I feel obliged to bring it in some detail before you. I refer to *Lupus;* and I comprise under that name not alone the common form, but certain other more or less rare varieties of the process, which I trust to be able to claim as certainly belonging to the lupus family. I am not about to trouble you with any detailed description of those several forms, but in order that the questions as to causes, relationships, and the diathesis behind them, should come clearly out, it is necessary that I should state briefly a few facts.

Concerning common lupus, then, I would assert that it is well known that it begins as a cell growth in the corium, that it always begins as a single patch, and always spreads at its edge by the infection of the adjacent skin, and that it very usually tends in the end to become multiple. Its multiplicity, as well as its local spread, is due to its infective power, and its secondary growths are almost always placed in close proximity with the parent one. It never in any case, however extensively multiple its patches may be, shows any tendency to symmetry; in all these features its clinical history resembles that of a local cancer. That which is true of the locally restricted form of epithelioma known as "rodent ulcer" is true also of common lupus. Both are slowly progressive new growths which after a time inflame and ulcerate, and then cicatrize. Both are curable by free measures of local extirpation and by those only. You cannot influence either except in the most minor degree by any remedies which appeal to the general health. Neither shows any power of producing gland disease, nor any tendency to spontaneous amelioration. The one is a disease chiefly, however, of early life, and the other of adult age. The varieties which common lupus pre-

sents do not offer us anything very exceptional to what I have said. According to the constitution of the individual, the state of health, the kind of diet, and the climate, lupus may inflame and ulcerate much or little, and in connection with these almost accidental differences, the old, and I trust now abandoned, terms *exedens* and *non-exedens* came into use. It may vary also very much as regards its restriction to a single original patch, or its tendency to multiplicity. Sometimes we see a single patch steadily spreading at its edge through the greater part of a long life, and never showing any tendency to produce new ones. This probably depends upon comparative vigor of constitution and a lesser degree of susceptibility. The explanation of the secondary patches when they occur is, in all probability, that they are the result of infection. As already stated, the multiplicity is never spontaneous. It is not, I think, beside the mark that I should ask attention to the methods of cure which have been proposed for lupus, as being indications of some affinity with the diathesis of malignant diseases. We have lately been taught by Professor Volkmann that every portion of the diseased tissue should be most carefully eradicated by the scoop. But his proposal had nothing whatever in it that was original in its theory; for long previously we had all been accustomed to act on the same principle, and to seek by the knife, the caustic, or the cautery, to utterly eradicate every fragment of the new growth. If I am not mistaken, we owe to Mr. John Gay—to whom surgery is indebted for many other practical suggestions—the radical proposal that the lupus patch should be cut out with the knife. It is essential that whatever method be adopted it be pursued unflinchingly and over and over again if needed. In this way lupus may be cured, and it is not too much to hope, now that this principle is becoming widely understood, and the profession is becoming bold in its practice, we shall before long witness a great diminution in the number of those who are disfigured by it. My object, however, in mentioning the success of vigorous local measures is not to enforce a lesson in therapeutics, but to intensify our conception of common lupus as a local new growth possessing infective properties. I might add that it often begins from some slight local injury, a sting, an abrasion, or a chilblain. Among the interesting varieties which common lupus presents we have a rare form, which

we may suitably call "acne lupus," which occur in scattered spots over the face, these spots being evidently connected with sebaceous follicles, and often with previously existing acne pustules. Their arrangement, however, I believe, never follows that of acne in being symmetrical, but always obeys the law of lupus, in that the secondary tubercles are more or less near to the parent one. In another variety, lupus attacks mucous membranes and produces slowly spreading ulceration, attended with papillary outgrowth. That lupus of mucous membranes as we see it affecting the conjunctiva, the gums, the palate, etc., is really lupus is proved by its very frequent association with well-characterized disease in the skin. In another extremely rare variety, of which I have seen but a few examples, the lupus growth is exceedingly trivial, does not ulcerate at all, spreads rapidly at the edge, leaving behind it a thin, supple, and healthy scar. In yet another variety it receives modification probably from the part in which it occurs. It is but seldom that we see well-characterized lupus on the hands or feet. But we often encounter a form of disease which takes its place, in which there is less evidence of new growth and more of inflammation and tendency to papillary hypertrophy. To this group, the form of inflammation due to dissection wounds on the hands—necrogenic warts of Dr. Wilks, or necrogenic lupus, I think belongs. There is yet another variety in which the surface of the lupus patch does not ulcerate, and is not raised, but weeps, like an eczema, presenting a condition which would at first sight, and certainly by the inexperienced, be mistaken for eczema. The facts to which we appeal, in proof that all these very different looking inflammations of the skin are really lupus, are that they all take origin at a single spot, spread slowly but constantly at their edges, and whether they ulcerate superficially or not, always leave a cicatrix where they have passed. I do not think that there can be much hesitation in accepting the statement, that a disease which presents these features, and is not either a cancer or syphilitic, must be a lupus.

We have arrived, then, at the end of our first stage in the inquiry, and have seen that this peculiar form of new growth in the skin and mucous membrane to which young persons are liable may, while observing identity in essentials, manifest very peculiar differences in external appearance. It may be in

single patches or multiple; it may look like itself, or like an acne, a lichen marginatum, or an eczema. These differences have more to do with the original differences in the texture of the skin which is attacked than any other class of causes. Let us notice the peculiar thinness and delicacy of the skin in the boy who furnishes us the example of what I have called *lupus marginatus*, in which exceedingly little growth and no ulceration occurred. We have now to take another step and to encounter another process with far more important differences. The diseases which are known as *lupus erythematous*, and its variety *lupus sebaceus*, are more than a mere modification of common lupus. They are distinct varieties, or, if one dared to use such a word, they are almost species. They manifest relationships with other type-forms of skin disease, almost as close as those they bear to common lupus. Had it not been for the fact that the common form and the erythematous form, often exist together or slide into each other by insensible gradations, we might have been tempted to prefer to call erythematous lupus by some other name. Its most important difference of feature is that it usually occurs symmetrically. Thus there is far more reason to believe that it is due to a constitutional peculiarity, than is the case with the absolutely non-symmetrical and local lupus. The arrangement of the bat's body and wings, on the nose and cheeks, in erythematous lupus is well known. The parts affected next to the nose and cheeks are almost always the ears, and next to them the backs of the hands. In this spreading there is no continuous connection between the patches. They are separate from each other, and apparently independent. The evidence as to multiplication by infection is far less strong than it is in the common form, though there can be not the slightest doubt that this kind of advance does take place at the edges of the patches. Remembering what I had to advance in our last lecture in reference to the possibility that all inflammatory processes may be capable of infecting the blood, I am unwilling to admit, even when we see lupus erythematosus on the backs of both hands, while the whole of the rest of the upper extremities and trunk remain free, that we have conclusive proof that these secondary manifestations are independent of the original patches on the face. The symmetry cannot possibly be due to any other cause than pre-exist-

ing proclivity of tissues; but it is just possible that it is not independent of an infective process also. At any rate, it is true that this disease is wonderfully symmetrical; the ears, the cheeks, and the backs of the hands show exactly the same conditions on the two sides. The variety to which the name sebaceous is given is often not erythematous at all. There may be no redness and no swelling, but the skin becomes dry, rough, and shrunken, and the orifices of the sebaceous follicles are very conspicuous, like the inner side of a nutmeg grater. This form is also accurately symmetrical, but it rarely leaves the face. If you see it on the nose you may look for it on the ears, and in the middle of the concha of each,—just where in acne a group of comedones is often seen,—there will be a little patch of lupus. Lupus sebaceous and lupus erythematosus are frequently mixed in the same case, and they are probably the same disease modified by peculiarities in the skin. Although I have described them both as usually symmetrical, not unfrequently exceptions occur. Under all conditions they show their alliance with lupus by leaving the skin which they have affected in a state of scar. I suggested that lupus erythematosus had alliance with other skin diseases, and I was thinking especially of psoriasis and those modifications of nutrition known as chilblains, and my suggestion will now be that it owes its peculiarities to the mingling in varying proportions of these two diatheses with that of lupus. As regards chilblains, it is sometimes, when the ears and the backs of the hands are affected, quite impossible to differentiate between them and lupus erythematosus. The latter, like chilblains, is always worse in cold weather, and it usually occurs to persons of feeble circulation, in whom venous congestion easily occurs when the surface is chilled. Its alliance with psoriasis is shown, not alone by its symmetry, but by the fact that in exceptional instances the scaly patches may be seen on the tips of the elbows, with now and then a diffuse psoriasis condition of the whole surface. Children and young persons are but rarely the subjects of lupus erythematosus, nor does it usually attack those in whom the skin is thick.

I think we may assert respecting lupus as a whole that it is a sort of cross produced by tendencies at once to scrofula and cancer, and that it receives many modifications from peculiarities in the patient's skin and his morbid tendencies

in one or the other direction. If he has a degree of tendency to the psoriasis diathesis it will assume the erythematous form, and it will be especially prone to attack the extremities if it encounter also the proclivities to chilblains.

I have yet left for consideration another variety of lupus concerning which a few words must be said. It is a disease which differs much from all forms which we have described, and yet I submit that it is really a lupus. I know as yet of but four recorded examples of it. The late Dr. Tilbury Fox showed at a meeting of the Pathological Society a young man on whose thigh were two different and very peculiar conditions—one being a congenital port-wine stain and the other a persisting vesicular eruption which had been developed at a later period. As a result of microscopical examination, Dr. Fox believed that the vesicles in question were in connection with the lymphatic trunks and spaces. At the same meeting I mentioned two cases in which I had for a considerable time taken much interest, but which we neither of us then thought to be exactly similar. A third case has subsequently come under my observation, and I have now no hesitation in believing that the four are examples of the same malady, and that the lymphatic trunks are really implicated. Whether it deserves the name of Lupus Lymphaticus I must leave to your judgment. All the four patients were boys; in all the disease had existed for several years; in all it was slowly spreading at the edge, and producing, near to the parent patch, secondary ones, which were apparently due to infection. In none of the cases was there ulceration, and in all the eruption consisted of little firm resisting vesicles which showed no tendency to rupture, and were aggregated into confluent groups. Upon these vesicles and at their bases were little tufts of capillaries which were almost nævoid in character. By microscopic examination of portions cut from all three cases, I have satisfied myself that the lymphatic trunks are dilated, and further, that there is a new cell-growth around them exactly like that of common lupus. So similar is this cell-growth to that of lupus that a very able microscopist, who examined one of my slides, took it for a specimen of that disease. It is right that I should add that in this microscopic examination I have been much assisted by Dr. Sangster, Mr. Robert Parker, and my eldest son. My belief is that the malady in question bears the same

relation to the lymphatics that lupus erythematosus does to the blood-vessels, and that it is an instance of a new growth assuming special clinical features in connection with the peculiarity of the special tissue in which it takes its origin. As in cancer everything depends on the tissue first affected, whether the growth is a melanosis, an epithelioma, or a scirrhus, so in lupus permanent modifications are stamped on the process according as it begins around the sebaceous glands, the blood-vessels, the lymphatics, or in the corium. When the type has been once taken it will be retained.

The various forms of *Iritis* are of peculiar value as indications of diathesis. Its occurrence in infancy, a very rare event, is usually indicative of syphilis. In adolescent periods, and early adult life, we have different forms of iritis, all of which generally imply the inheritance of gout. This inheritance is, in such cases, often modified by other individual peculiarities, such as feebleness of circulation and proneness to chilblains. When this is the case, a very peculiar form of multiple arthritis which begins in the last joints of all the digits, and often affects them exclusively, is sometimes seen. The explanation of last-joint-arthritis is doubtless to be found, as I have just suggested, in the peculiarity of circulation which in later life gives tendency to symmetrical gangrene of the extremities, as so well described by Raymond, and more recently among ourselves by Dr. Southey. It is not, however, due to this alone. There is always joined with it an inherited tendency to gout. The form of recurrent iritis which we meet with so frequently in adult men, but occasionally in women also, in which the attack is sharp and severe, the interval of immunity complete and the recurrences take place once a year, or oftener, is, I believe, distinctly an indication of rheumatic gout. It does not very often happen to those who suffer severely from declared gout, and it is very rare in connection with rheumatism only. We meet with it in those in whom there is inherited proclivity to gout, who have lived rather freely, who are susceptible alike to weather and to diet, but who have never themselves had an outbreak of true gouty arthritis. The fact that this form of severely paroxysmal iritis occurs much more frequently in men than in women, supports the general statement which I have just made. In women of middle age, or a little past it, we encounter iritis in

another form, as an insidious disease, attended by a little conjunctival redness, but with some sensation of heat and irritability, and gradually, without any definite paroxysms, producing extensive adhesions. The little exacerbations which attend it are often more definitely influenced by exposure to cold and wind than by diet. This form of iritis is, I believe, almost invariably a consequence of a remote inheritance of gout.

I tried in a former course of lectures to give a definition of the word *Catarrh* which should enable us to employ it for more accurate clinical purposes than it is often used. I suggested that it should be held to imply identity in cause, and not similarity in result. There is surely no good reason why all forms of inflammation of mucous membrane, attended by free discharge, irrespective of their causes, which are often very different, should be called "catarrhal." On the other hand, if we use the word in the sense which I contend for, we shall find it applicable to other inflammations besides those of mucous tissues. In so doing we shall construct a large and perfectly natural group of maladies. Whatever is the result of catching cold is catarrhal; conditions which follow from other and dissimilar causes, however similar their results, ought to be refused that name. The catarrhal diathesis, using the word in this sense, is one of the three fundamental ones. It is not due to any specific or any specialized cause. Every organization possessing a nervous system must be supposed to be capable of manifesting it, for its essence consists in proneness to inflammatory congestions excited, in a reflex manner, through the influence of cold applied to the surface. The susceptibilities of the nervous system, however, in this direction differ, as we all know well, very greatly in different individuals. These differences are hereditary, and may easily become the possessions of families or of race. Not only do individuals differ in the degree of reflex susceptibility, but they also vary much as to the special tissues or organs which are most prone to suffer under it. Thus, some catch cold, almost solely in the mucous membranes of the nasal passages and pharynx; in others, the tonsils, throat, and larynx are more prone to suffer; in others the bronchial mucous membrane; and in others the stomach and bowels, or even the liver. Although I shall admit rheumatism to the rank of a

universal diathesis and place it for the present in this respect side by side with scrofula and catarrh, yet in doing so I shall have to suggest, as indeed I have repeatedly done, that rheumatism after all is only the catarrhal diathesis affecting the joints. Let me state a few general facts respecting catarrhal affections, and then pass on. First, it is a great mistake to imagine that only those of delicate constitution, and accustomed to much protection from weather, etc., are liable to catch colds. Farm laborers and others engaged in laborious out-door pursuits, often show this susceptibility very definitely, and suffer very severely. My impression is, however, that the peculiar degree of susceptibility which causes its possessor to experience slight catarrhal symptoms on every little variation of accustomed exposure, is rarely reached in them. They take cold but seldom, and when they do, it is in connection with some exceptional cause, and the catarrh developes with exceptional severity. I have, however, met with some remarkable instances of extreme development of the catarrhal diathesis in those who are constantly out in the open air. Especially well I recollect an apparently robust farmer, who could do anything he liked out of doors, but who could not sit in a carriage with an open window, or for ten minutes with his feet on a stone floor, without catching cold. It is a general law of all catarrhal affections, and well recognized in the experience of every one, that they are spontaneously curable, but very liable to recurrence.

All forms of catarrh pass off spontaneously after a while, and in many persons their duration is tolerably regular, and the period of immunity often keeps to the same average length. When we find the results of a catarrhal inflammation threatening to become lasting we feel sure that the diathesis is a complicated one. It is one which very easily mixes itself up with other forms of ill-health.

We define scrofula to be a state of the solid tissues, and more especially of the lymphatic system as a whole, in which there is peculiar proneness to chronic inflammations resulting in products more or less peculiar and specialized. Modern discoveries will perhaps lead us to believe that those specialized products are usually accompanied by the presence of particulate organisms which are neither part of, nor derived from, the original tissues. Be this as it may, we shall have

to study the scrofulous diathesis, and the predisposing causes of scrofula, just as before. We shall still have to ask what are the conditions which favor, or otherwise, the development of the bacillus in question. Even the firmest believers in the new facts admit this. Looking, then, upon scrofula as a permanent and heritable condition of the tissues favoring chronicity in all inflammatory processes and directing them towards more or less specialized ends, it is easy to see that in such individuals catarrhal attacks may depart from their usual type, and may not show their natural tendency to spontaneous disappearance. As a matter of fact, we are only too familiar with the danger of catarrhal attacks in scrofulous subjects.

But it is not alone in the scrofulous that we encounter risks in connection with catarrh. Those in whom the tissues have undergone senile degeneration, and those, too, who although not old have become seriously enfeebled by other causes, often show an inability to shake off catarrhal attacks, and a similar result sometimes brought about under the law of habit, when catarrhal attacks have been exceptionally frequent and severe. Apart from the risk of lapsing into chronic disease, catarrhal inflammations are in themselves not unfrequently dangerous to life. Many of the forms of acute bronchitis and pneumonia, and some forms of enteritis, peritonitis, and pleurisy, are distinctly catarrhal in their nature—that is, they result from the exposure of an individual to the ordinary causes of a catarrh—and in many of these the popular expression is verified, "he caught his death of cold." Rheumatic fever very often, as is well known, acknowledges a similar cause; and respecting it and the other acute and dangerous catarrhal inflammations, one general statement is true—that if the patient can only live through the attack it will in due course pass off, and pass off completely, leaving him again in good health. This remarkable facility of spontaneous recovery which is a feature of all catarrhal, or, in other words, of all reflex nervous inflammations, has placed physicians in great difficulty in coming to any satisfactory conclusions as to the relative merits of different plans of treatment. I will not say that cases of acute pneumonia, or acute rheumatism, or severe catarrh recover as well under one plan as another—I should be going far out of my province to make any such insinuation, and I should be saying, too, that which, with my small amount

of information, I certainly do not believe. This, however, I must repeat—that there is in each and all of the examples of this type of inflammatory attack, a natural tendency to recovery at a certain stage, which the student of therapeutics must always keep in mind. Although I have spoken of cold, in combination with damp, as if it were the sole cause of catarrh, it must by no means be supposed that this is exactly so. With the changes in temperature there are doubtless, frequently coincident and very influential, certain electric disturbances, of which it is very difficult to take exact appreciation. To these we must refer the great prevalence of catarrhal and catarrho-rheumatic affections at certain seasons, and during the prevalence of certain winds. This prevalence is sometimes such as to suggest, as in the case of influenza, that the air must carry with it particulate and contagious germs. The sum total of what are included in the meteorological conditions termed weather must count as causes of catarrh.

I have placed the rheumatic diathesis with the catarrhal and the scrofulous, as one which is universal, and in which all human beings share in some degree. What I have said as regards the laws of catarrhal inflammation apply very closely to those which are rheumatic, for the two are, as regards their causes, very nearly allied. Rheumatism like catarrh is usually transitory, especially when it occurs in the young. As with catarrh, so with it, when the aged are its subjects there is danger that it may become chronic or even permanent. Like catarrh, it may mix itself with scrofula or be modified by any other influence which disturbs the general health. The effect of malaria upon the system is often seen to much increase the proclivity both to catarrh and rheumatism. One other diathesis so very frequently mixes itself up with rheumatism, and the two in hereditary transmission become so intimately united that it is a matter of considerable difficulty to ascertain how far rheumatism pure can go. We can, however, have little hesitation in admitting that it may be the parent not only of rheumatic fever, of transitory rheumatic pains in joints, fascia, and muscles, but of chronic crippling arthritis, in which almost all the joints are involved, and in senile periods of the slowly destructive arthritis which wears away the cartilage and eburnates the bones. Further, I think

we may grant that it is not unfrequently the parent of sciatica and lumbago. All the affections which I have mentioned occur occasionally to those in whom there is no reason to suspect a gout complication; and, what is a still stronger fact, they are met with occasionally in the lower animals. Having said this I must not delay to add that in a great many of the exaggerated and peculiar forms of them which we witness in the human subject, the cause of the peculiarity is an hereditary complication of gout. When this complication exists it shows its power, we may suspect, by inducing a permanent modification of tissue; and it is to this modification that the peculiarities in the processes are due. Hence the impossibility, under many conditions, of discriminating between gout and rheumatism. We can for the most part easily tell, in any individual case, what the causes which have provoked the attack have been, and we can assign them on the one hand to those of rheumatism or on the other to those of gout; but we cannot estimate with any chance of certainty the previously acquired proclivities of the tissues. There is yet another source of fallacy, that we are by no means always acquainted with the condition of the patient's health at the time of the attack. If a patient already predisposed to gout, in his kidneys, his tissues, or his blood, be exposed to the exciting causes of rheumatism, he will have one of those attacks which you yourself, Mr. President,[1] have so well described, and about the nature of which there has been some controversy. There will be difference of opinion as to whether such an attack should be called rheumatic fever or generalized gout; and the solution of the question is, I suspect, to be found in the fact that it is both. Two sets of causes have been at work, and the result has been a mixed one.

When a little white tophus is seen on the ear, the merest tyro can read its lesson, and the veriest sceptic dares not refuse its revelation. It is both retrospective and prospective in its significance; we know that the patient's kidneys have become unsound, and that he has failed, in the past, in the due depuration of his blood. We know that this loaded blood condition must have put him in danger of attacks of arthritis; and that in all probability he has suffered on many occasions from

[1] See Spencer Wells on Gout, p. 66; also Garrod on Gout, p. 42.

acute and very painful inflammations of some of his smaller joints. We know further that changes have taken place in many of his tissues, and especially that the walls of his blood-vessels have become thickened. Looking into his future we know he is in danger not only of other attacks of arthritis, but that, however carefully he may restrict his diet and manage his mode of life, it is probable that the renal and arterial changes will increase and bring with them their peculiar inconveniences and risks. We know further that if a man in such a state should have children, they will, in spite of the utmost care in avoiding the dietetic causes which originally produced it, inherit some of the peculiar tendencies which he had acquired. Where the inheritance will stop, how far it is possible for one structural peculiarity to be isolated, and to be inherited apart from others, we do not know. There is, however, I would suggest, good reason for believing that in families in which, either by accidentally changed conditions, or by intelligent foresight, several generations have been carefully exempted from the causes of gout, its existence, in times long past, may still be shown in the perpetuation of one or other, more or less isolated, tendency to structural disease. It is under such conditions that not a few of the forms of rheumatic gout occur, although in others, and perhaps a large majority, it is rather the fact that the exciting causes of gout have by no means been wholly suppressed. In this way we may have to recognize, as lineal descendants of gout, maladies which occur in those in whom we can by no means prove its persistence. I have referred repeatedly to the facts which favor the belief that the hæmorrhagic diathesis itself may, perhaps, belong to this category; and in a former course of lectures I analyzed the facts contained in Dr. Wickham Legge's able monograph on this subject. During the past year some very interesting cases have come under my observation, which serve to give support to it, and to lead to the conclusion that many, if not most, forms of intractable hæmorrhage are in connection with hereditary peculiarity of the arterial system, derived from gouty progenitors. I will mention only three, and these very briefly. A gentleman who consulted me a few weeks ago, with cataract and diabetes, told me that he had been most of his life liable to very profuse attacks of hæmorrhage. In early life they had been alarming, and sometimes

so frequent as to be almost constant. They were always preceded by a sense of weight and discomfort at the epigastrium; and provided the hæmorrhage was not too profuse he had usually felt relieved by it. There was a strong inheritance of gout, and he had himself suffered from several attacks. A school-boy was sent to me by Mr. Harrison, of Braintree, on account of repeated and alarming attacks of epistaxis. They had been so profuse and so frequently recurrent that he had lost much of his color and strength. The point, however, which most caused anxiety was that his elder brother had died of it. In him the liability had commenced at thirteen and a half, had persisted for two years, and finally, after continuing for four days, it ended in death. Their paternal grandfather had suffered much from gout, and their father at the age of twenty-two had had severe epistaxis. In the case of a lady, now aged twenty-nine, and much out of health, I had the following history:—She had always suffered from indigestion and rheumatic pains. At the age of seven she had epistaxis to such an extent that the nostrils had to be plugged; and in spite of that measure it was, during three days, expected that the attack would end fatally. She once had bled to an alarming extent in consequence of tocth-drawing. Her first menstruation had soaked the bed through; and ever afterwards this function had been attended with losses so profuse as to have greatly enfeebled her. There had been much gout on both sides of her family, and she herself had had symptoms very like it.

I was unable, when examining this subject last year, to agree with Dr. Wickham Legge in his opinion that cases of remarkable tendency to hæmorrhage, occurring, as it were sporadically, should be kept quite apart from the hæmorrhagic diathesis, in which we observe the tendency to prevail in direct descent through many generations. It may be that as a matter of convenience it may be well temporarily to effect this separation, but it must, I think, be with the clear understanding that it is a conventional one. The hereditary cases are those in which the idiosyncrasy has become definite and established; the others show us the idiosyncrasy in connection with its original causes, and, if I may so express it, in process of breeding. The cases of hæmorrhage into the vitreous in young men suffering from constipation which have been

collected by Mr. Eales, of Birmingham, and in many of which no history of gout could be obtained, go with other facts to make it probable that tendencies to bleed are sometimes met with in association with other causes. My own experience as to retinitis hæmorrhagica, blood patches in the conjunctiva, cerebral hæmorrhage, epistaxis, and other allied disorders, leads me, however, to attach very great importance to the history of inherited gout as their frequent cause.

There is one subject which it is impossible to leave out of the account in our estimation of the diathesis. It does not constitute a diathesis in itself, but it complicates them all. I refer to the changes in the state of the nervous system, or quality of the nerve force, which we know as *tone*. By that word I suppose that we mean the power of producing nervous energy. If we speak of the tone as low and poor, we mean that there is but little vigor in resisting irritation, and that comparatively slight influences will evoke a large amount of disturbance. That vigor depends, no doubt, upon the state of nutrition of the nerve-cells themselves, and it may vary much in different parts. Thus, the tone may be diminished in one nerve district and yet be good in others, while in some instances it is reduced in all, simultaneously. A condition of great general exhaustion is by no means, however, always equivalent with a state of low tone; it is necessary that the enfeeblement should pertain to the nervous system especially, in order that that term should become applicable. Perhaps it is further true that in most of the conditions in which derangement of tone is in question, the disturbance or enfeeblement has reference chiefly to that part of the nervous system which controls the circulation. Whenever the circulation is specially liable to reflex derangement, and congestions or their opposite occur with unusual facility, we are safe in declaring that the tone is low. It is part of the business of the nervous system to duly regulate the supply of blood to various parts, to prevent local arterial spasms, and under the varying conditions of daily life to maintain the normal balance of circulation. When this power is deficient the various causes of disease act with greatly increased effect. In many cases loss of tone may be so long continued and so great that we cannot but suspect that it depends upon degenerative and permanent changes in the nerve cells. Not only may loss of tone aggravate a dia-

thesis, but many of these act very efficiently in producing loss of tone. Thus the influence of malaria is very definite in this direction, and is felt through very long periods. But I cannot for the present dwell longer on this part of my subject.

The present is, perhaps, the most suitable place for me to interpolate two emendations on former lectures. In my first lecture I adverted to the want of observations for the purpose of estimating the relative frequency of the different complexions in the British population. In doing so, I omitted to mention the excellent work accomplished some years ago by Dr. Beddoes, of Bristol. I had intended to mention it, and should certainly have done so if I had remembered its extent and value. In a subsequent lecture I urged the importance of Life-Records, which should be kept in advance, ready to be produced to any medical man who may be consulted, and especially comprising mention both of family tendencies and of idiosyncrasies of the individual, and the diseases which have been passed through. In making this suggestion, which had long occupied my thoughts, I was not aware that the Collective Investigation Committee of the British Medical Association already has in hand a scheme for its realization. I have since with great pleasure been informed that such is the fact.

It is time that I should bring this lecture to a close, and with it will end my present course. In doing this, I desire, gentlemen, to thank you most sincerely for the very courteous attention with which you have listened to what I have advanced. Permit me at the same time to attempt a very brief *résumé* of the principal arguments.

We began by a glance at the praiseworthy and to some extent successful attempts made by our forefathers by looking into a man's face to determine therefrom his physical peculiarities and his liabilities to disease. On examining the facts supposed to indicate "temperament," I was obliged to conclude that part of them were merely the characteristics of different races; and another part, merely the products of past disease—personal or inherited. Thus it seemed that if race took its share, and disease that which legitimately belongs to it, there was very little left to be classed under the head of "temperament." As regards diseases in general, I suggested that the plan most likely to be productive of good results is to classify the chief groups of morbid influences, and to endeavor

then to associate with each the separate maladies and tendencies which are dependent upon them. To this object our last three lectures have been devoted. We had previously endeavored to clear the ground somewhat by placing in a separate category a large number of symptoms and of morbid manifestations which take their origin in individual peculiarity, quite apart from any recognizable tendency to disease. I insisted that the domain of mere *Idiosyncrasy* is very large. I suggested that these peculiarities may have had their origin remotely in disease, but asserted at the same time that they have often existed for many generations in complete freedom from any such association. Whether I have succeeded in convincing you that it would be a convenience in medical teaching and practice to revive the old doctrines of diathesis I do not know. To my own mind it does appear, I may confess, that the states to which I have sought to apply that name are realities which it is very desirable to recognize. It seems to me, also, that the habit of naming diseases as if they constituted separate and complete entities has often a narrowing effect upon our pathological conceptions, and leads to the overlooking of special causes. My endeavor has been to look at disease not exclusively from before, but, so to speak, to get behind it and trace its origin. Classification should surely depend not upon external similarity, but relationship in cause. We should seek to place diseases in natural groups, in connection with their ancestral descent. The attempt to do this has, I fear, sometimes led me into speculations as to the genesis of the different forms of diathesis which may have seemed somewhat transcendental, and been possibly a little wearisome. I can only plead that I have done my best to avoid these faults, and I have been endeavoring to thread my way through the mazes of a most complicated topic. The main conclusions as to the diathesis which I have suggested for your acceptance are these:—That there are three great universal diatheses dependent upon the very commonest cause of disease by which man, and not man only, has been assailed from the most primeval times. These are—the catarrhal, the rheumatic and the scrofulous. Close to these come others of far less importance, but of parallel nature, because they comprise us all in their range of liability; the diathesis of senile degeneration and the diathesis of malignant new growths.

Next we have certain very important ones which are widely spread, but by no means universal, since they depend upon local exposure or personal habits. In this class we have the ague-diathesis, the diathesis of rickets, those of bronchocele, scurvy, leprosy, pellagra, and gout. Respecting the diathesis due to *malaria*, I have some doubts whether it ought not to be admitted to the dignity of a primary or universal diathesis. If we go far enough back, probably the whole human race has been submitted to its influence, and not less probably has some impression from it been stamped upon us all. It may even be doubted whether now, in our advanced stage of civilization, those who are apparently exempt from all possibility of influence from marsh miasm may not still, in some minor degree, suffer from its remote effects. It is very possible that, although the severity be far below what is needed to produce an intermittent, yet that the well-recognized ill-effects of certain winds quite disproportionate to their chilling power may be due to slight impregnation with malarial poison. As to the diathesis of gout, I have suggested that it is but rarely of pure breed. Very often it builds on the foundations laid by other maladies, and however much it may boast itself *dominus morborum, morbus dominorum* it is, if we see it aright, very often merely a complication of plebian rheumatism. Other diatheses have been mentioned which it is difficult to place in any definite arrangement. That in which there is remarkable tendency to hæmorrhage I have associated, for the most part, either directly or remotely with the disease of the arterial system induced by gout. That which is characterized by failure of nerve tone I have linked in a like manner —not invariably, but still very commonly—with the influence of the sexual system upon the general health. So also of the state characterized by undue mobility of nerve force, or emotional susceptibility, known as hysteria. That there are hereditary diatheses which are characterized by peculiarities in the state of the different viscera, or of specialized systems, has also been suggested. In this category we have the hepatic diathesis, the diathesis denoted by chilblains, and that in which the vascular system generally is very feeble; and there results the liability to venous congestion in exposed and distant parts—even to gangrene of the extremities. I made no attempt at completeness in the enumeration of the diatheses, for

it is clear that a little ingenuity might subdivide or define them to an almost endless extent. The description of a fresh diatheses is, in fact, almost as easy as the discovery of a new nerve centre, or the revelation of a new bacterium. Let me not be thought to speak in the least slightingly of modern investigations in the two latter directions. I simply couple them with the attempts made to classify morbid phenomena, and to say of them all that they are, for the present, tentative, and that the experience of the profession in the future must assign them their real value.

Finally, I think I may claim in all that I have said as to idiosyncrasy and diathesis, that I have done my best to recognize the power of hereditary transmission, and to insist that we ought to study disease as being, not of recent origin, or in dependence solely upon existing influences, but rather seek to truthfully "read the record of its long descent."

Again I must ask pardon, as I have already once done, for the fact that I have doubtless often appeared to be stating as somewhat novel, things which are well known and almost commonplace. I could not possibly, from the nature of my subject, have avoided this, nor, indeed, have I made much attempt to mark out what I wished to claim as original. It is abundantly sufficient for my ambition if, availing myself thankfully, so far as my knowledge extended, of the labors of those who have gone before me, I have succeeded in any degree in making opinion more definite, and giving emphasis to that which is true.

LECTURES ON THE TREATMENT

OF THE

Common Diseases of the Skin,

BY

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COMMON DISEASES OF THE SKIN

LECTURE I.

PRURITUS.

BEFORE considering the treatment of skin diseases, it is advisable to refer to *Pruritus*, a condition common to many of them, and to nearly all of those we meet with in ordinary practice. No symptom is so troublesome to the patient, or more readily brought under the influence of remedies, provided the cause can be ascertained. Such a cause is furnished by any irritant, an animal parasite for example, which induces hyperæmia and stasis of the blood in the papillary body. In consequence of this the terminal filaments of the nerves are pressed upon and itching results. It is, however, important to remember that general itching may result from localised causes, and hence, a single louse may be the cause of diffused and intolerable Pruritus.

The presence of the itch insect, of bugs or fleas, and less frequently of vegetable parasites, especially the Tricophyton Tonsurans are common causes of itching.

Eczema is essentially an itching disease, especially during the acute stage; and psoriasis also induces itching, but in a lesser degree than eczema. In both of these diseases the itching is aggravated by warmth, and is especially complained of when the patient is warm in bed.

Syphilides, as a rule, itch very little or not at all, and only on their first appearance; hence, this fact is one of very great importance in making a diagnosis between simple and syphilitic eruptions.

When Pruritus is due to any of the above causes, it may easily be relieved, and cured entirely by the removal of the

exciting cause, but when it is so to speak a substantive disorder, and the first symptom of skin trouble, local or universal, its treatment becomes difficult, and often very unsatisfactory.

Such conditions are pruritus ani, and pruritus senilis.

Pruritus ani is often due to eczema, thread worms, piles, or excessive sweating, but occasionally occurs independently of these, and is frequently associated with a simila- affection of the scrotum in men, or the vulva in women.

Pruritus senilis is possibly due to trophic changes in the skin, especially the loss of subcutaneous fat, and is most difficult to treat. Like all other forms of pruritus it soon becomes associated with the presence of excoriations. These occur naturally on those parts which are most easily reached by the fingers, and especially about the lower part of the body and the shoulders.

As might have been anticipated, pilocarpin and hot baths, which increase the activity of the sweat glands, are harmful, and the greatest relief is afforded by cold bathing, and by the use of evaporating lotions. Preparations of tar and sulphur are generally recommended, but in my hands they have availed little; sedatives such as opium and potassium bromide are of no curative value. It is possible, of course, to get a good night by means of narcotics in large doses, but the disease is always chronic, and the abuse of narcotics, which at best procure a brief respite, is a remedy worse than the disease. Very dilute solutions of hydrargyri perchloridum (gr. xv. to a pint of lime water) sometimes relieve, but I know nothing better than the free use of Eau de Cologne, or of Hazeline, mixed with an equal amount of cold water. These should be applied with a sponge, and frequently used, especially before going to bed. In some cases I have thought relief has been afforded by taking diuretics, and so throwing part of the work of the skin on to the kidneys. The acetate of potash in twenty-grain doses answers this purpose admirably.

If pruritus ani be due to hæmorrhoids or worms, the treatment of the symptom becomes that of the disease.

Serious hæmorrhoidal trouble of course necessitates drastic and surgical treatment, but the milder form will generally improve under the use of unguentum gallæ copio, or unguentum hydrargyri. The treatment of thread worms is familar to all of us, and its non-success, unfortunately, only too fre-

quent; but I have latterly had surprisingly good results from the internal administration of lime juice, after the failure of the ordinary remedies. If pruritus depends on excessive sweating, the parts should be dusted with a powder of equal parts of tartaric and boracic acids.

If it appears to be idiopathic, an ointment of equal parts of oleate of zinc ointment, and unguentum picis liquidæ will prove useful, and in some cases I have added, with advantage, four grains of cocaine to an ounce of the ointment. Pruritus vulvæ should always excite the suspicion of Glycosuria; it is by far more frequent in the Glycosuria of old adults, than in the declared Diabetes Mellitus of younger women. When due to this cause, or to Leucorrhœa, extreme cleanliness is, of course, to be advocated, and it will generally be relieved by the use of proper internal remedies, and the application of a lotion containing borax:—

℞—Glycrini Boracis ℥ ij
Aquam ad ℥ viij
M ft. lotio

One case, which had long resisted all means devised for relief, got well at once when the vulva was dusted with calomel. In this case, however, there was no diabetes or leucorrhœa or any suspicion of syphilis.

The itching of eczema may generally be relieved by the free use of lotions, containing hydrocyanic acid, 3 to 6 minims to the ounce, or liquor carbonis detergens, 1 to 2 drachms to the pint of lime water, but the greatest relief may be obtained by free sponging, with a decoction of bran. The bran should be boiled in a pudding bag, and the liquor, mixed with warm water, be freely used. In some cases lotio nigra, diluted with an equal quantity of lime water, has proved of great service.

Scabies is a cause of very severe itching, and its treatment often keeps this up. After the acari have been destroyed by the use of sulphur, it should be discontinued, as, if persevered with, its use often developes and keeps up eczema, and the itching dependent thereon.

There is another class of cases in which itching is a prominent symptom, such as icterus, urticaria, and scarlatina, in this last occurring with the outbreak and disappearance of the exanthem.

It has not been my experience often to meet with it in simple jaundice, but several times I have seen cases of gallstone colic, with or without a moderate amount of jaundice, in which the colic has been followed by intolerable itching. One lady, after enduring the misery of sleepless nights, and irresistible temptation to scratch, with the usual result of sores and excoriations, consulted me in the hope of obtaining relief. By the aid of bran bath and the lotio nigra, she obtained some respite from her sufferings, but it was not until she was given half drachm doses of liquor hydrargyri perchloridi and drachm doses of succus taraxaci that she began to make definite progress, and ultimately recovered.

In this case there is no doubt that the presence of bile acids in the blood was the cause of her trouble, but what the morbid agent in urticaria may be is not so well known.

No doubt in the great majority of cases urticaria depends on the presence in the stomach of foods that, ordinarily innocuous, cause the disease in individuals predisposed thereto. The obvious indication is to get rid of the offending material by means of emetics and purgatives, and subsequently to give vinum antimoniale for a few days, in doses of from 3 to 7 minims. Most cases will yield readily to this treatment, but there are others, depending not at all on digestive disturbances, which are most intractable. These usually occur in people of a nervous temperament, and in women seem to be sometimes associated with uterine disturbance.

One lady, herself very nervous, and having a strong family disposition to nervous ailment, such as hysteria and migraine, was brought to me after having for seventeen winters been rendered unfit for all social obligations by the most terrible urticaria. She had been obliged to spend several winters in the south of France, but no medicinal means had availed her anything. The use of hot bran baths and antimonial wine did her much good, but she still remains liable to the disease. Lotions of dilute hydrocyanic acid, three drachms to a pint of lotio nigra, and of hazeline, five ounces to a pint of water, have seemed to do good in some of these cases, but they are very difficult to treat, and tax all the resources of medicine.

LECTURE II.

ECZEMA—ETIOLOGY.

ECZEMA has commonly been defined as a catarrhal inflammation of the skin, but we attribute such a definite meaning to the word catarrh, and associate it so generally with diseases of mucous membranes, that its application to a widely different disease of the skin is scarcely correct.

We are then driven back to the unsatisfactory necessity of having to speak of eczema as being due to an eczematous inflammation of the skin.

Pye-Smith summarises the pathological anatomy of eczema as follows:—"If a section of eczematous skin be made, the cuticle is found unaffected, the malpighian layer swollen, the papillæ œdematous with dilated blood vessels, and multitudes of leucocytes clustered round them; the deep layer of the cutis and subcutaneous tissues are unaffected."

The prime factor in every eczema is dilatation and congestion of the superficial blood vessels, and upon the amount of this congestion, and on its secondary effects, depend all the varieties of the disease. In skin diseases, in common with many others, the tendency has unfortunately been to multiply names unnecessarily, and the true connection of many conditions has thus been obscured.

Of eczema this is especially true, but fortunately practitioners have ignored names, and treatment has thereby not suffered so much as might have been anticipated.

The congestion may never pass into exudation, and redness may be the only sign of disease. This we call erythema, or eczema erythematosum, and often enough, even without treatment, it may pass away leaving only a little desquamation of the cuticle to tell its tale. Beyond the redness nothing is complained of but a little itching and burning, and if erythema did not often pass into other forms of the disease, it would hardly seem entitled to rank as an eczema.

We see, however, exudation following the congestion, and further delevoping tissue change, and inflammatory activity of the connective tissue corpuscles; and on the extent of these further changes depends the particular variety of the disease.

Sometimes the erythematous stage is succeeded by the appearance of little red dry elevations called papules. These again frequently remain unaltered throughout their existence, and constitute the disease we call Lichen, or more properly eczema Lichenoides. Their summits may, however, be converted, and finally the whole bulk of them, into vesicles, and the affection then become the common form of the disease known as eczema vesiculosum.

This may seem to begin with a crop of vesicles at once, but there has always been an erythematous stage, however short.

The thin epidermal roofs of the vesicles soon break down from attrition, or from becoming thinned by the pressure of the contained fluid, and we get the characteristic eczematous discharge, the saline constituents of which act as irritants, while its albuminous part stiffens linen, clothes, or bandages in contact with it.

If the eczema is one of a pustular nature, it may follow on the vesicular form, the bases of the vesicles becoming inflamed and their contents pustular; but often the exudation is at once, or nearly at once, purulent, and we get the disease known as impetigo, or more properly eczema impetiginodes.

The pustules, just as do the vesicles, soon break down, and the pus dries into crusts which characterise this form of the disease.

It is common, however, to find all these forms of disease co-existing, and we rarely see a well-marked case of eczema, in which we do not find all four varieties simultaneously.

Besides these four principal varieties of eczema:—

1. Eczema Erythematosum,
2. Eczema Lichenoides or Papulare,
3. Eczema Vesiculosum,
4. Eczema Impetiginodes or Pustulosum,

we get other specially named kinds, which depend entirely upon the course and duration of the four principal varieties, and are called from the appearances they present, eczema madidans or rubrum, eczema squamosum, eczema rimosum and eczema verrucosum.

Eczema madidans or rubrum may result from any of the four chief varieties.

Duhring describes it as being "characterized by a more or less reddish weeping surface, accompanied by marked inflammatory symptoms. Serum exudes freely, and at once forms into crusts; blood likewise oozes forth from the lacerated and exposed corium, which, together with the serum, dries into thick, bulky, dark yellow crusts, completely enveloping the region. These crusts adhere closely and firmly to the part, and unless detached by mechanical means, may remain there indefinitely, the disease continuing its course beneath this mass of effete matter.

"Eczema madidans then may present two appearances,—as it occurs with its crust, and as it exists without this covering. In the one case the skin itself is altogether obscured by a dirty yellowish or brownish crust; in the other the skin presents a bright red punctate wounded surface, deprived in great part of its epidermis, and exuding a clear syrupy yellowish fluid."

It occurs most commonly on the legs, especially of elderly people. It is generally chronic, and involves the deeper tissues, the skin becoming almost leathery.

Eczema squamosum, rimosum, and verrucosum depend upon the amount of thickening of the corium and the rimosum or fissure-type upon its situation, cracks occurring over the flexures of the joints, especially of the fingers, owing to inevitable movements. They present a red scaly surface, and are always associated with a great amount of exudation, which converts the skin into a thick inelastic mass. So long as this thickening persists, the disease cannot be considered as cured, and though the surface may appear normal, any slight disturbance will occasion a fresh outbreak, unless absorption of the exudation has taken place.

It will greatly facilitate the diagnosis and treatment of eczema, if we regard these forms of disease as variations from a common type, rather than as separate diseases.

We have seen that the four principal varieties may present their peculiar characteristics practically from the first, yet they do more often lapse from one into another, according as the inflammation is more or less acute.

Allied as they are pathologically, so are they by their clinical features, which may be summarised as follows:—

1. Unusual tendency to persist.
2. Invariable presence of itching.
3. The fact that when cured they leave no scar.
4. The absence of contagious properties.
5. The evidence of heredity.

A favorite pursuit of dermatologists has been to search for a satisfactory classification of skin diseases, and eczema has, by first one and then another, been classed under this or that head.

The latest writer on the subject, Mr. Tom Robinson, has, I think, wisely and boldly spoken of the eczematous diathesis; but after all it is only a change of name, and instead of the herpetic, or the dartrous class of the French, or the catarrhal diathesis of Hutchinson, we simply speak of eczematous patients as having an eczematous diathesis; but when we come to consider the evidence of this diathesis, we find a list of symptoms alarming, it is true, but not sufficient to include all or nearly all those who have had or may have eczema.

Nor is it probable that we shall find a classification for a disease which varies so much according to its cause, and the constitution of the patient who is suffering from it. No doubt some people are specially liable to suffer from eczema, but there are certainly no sufficient data for predicting of an individual that he has, so to speak, "The mark of the Beast," and will suffer from eczema. It is true the eczematous diathesis is made to cover a wide area, and includes those who in early life are prone to chilblains, to a catarrhal condition of the mucous membranes, and to the advent of some form of irritation of the skin during cold weather.

Diathesis or no diathesis, some people get eczema from causes insufficient to affect others, and these causes are either internal or external; for eczema never occurs idiopathically, as I think it should if it were the manifestation of a diathesis.

Hebra calls the internal causes unknown, and the external causes known. This may be a witty aphorism, but is neither fair nor correct; if it were so, and we had no knowledge of internal causation, we should be powerless to prevent a patient who had once suffered from eczema from a repetition of the evil.

It is difficult indeed, but just as in urticaria we can, by regulation of the diet, often prevent a recurrence, so can we in

a gouty subject prone to eczema, by careful dieting, by the exclusion of alcohol, and the medicinal treatment of the gout, check the tendency to a relapse.

Again, a strumous child who has once had eczema will surely suffer again if his health becomes impaired, no matter how closely he be watched, and how thoroughly irritation of the skin be prevented. We know well enough that general hygienic and medicinal treatment will nearly of a certainty prevent an outbreak.

The known causes are such as produce irritation of the skin, and common among them are parasites, vegetable or animal, especially ringworm, which sets up an eczema that often masks the disease, the presence of dirt, and some medicinal remedies, notably chrysophanic acid and mercury.

Some skins are so tender that flannel will produce an eczema, and for these silk undergarments will be found an efficient substitute.

It must not be forgotten that eczema is not unfrequently set up by the use of badly-dyed underclothing, in which the aniline dyes contain an excess of arseniate of alumina and act as irritants.

People predisposed to eczema usually suffer from it when they are much exposed to cold east winds, and sea air will almost certainly rekindle a half-cured eczema.

Lastly, certain trades, by which patients are much exposed to contact with sugar or flour, will produce special kinds of eczema which are known as bakers' or grocers' itch.

LECTURE III.

ECZEMA: CONSTITUTIONAL TREATMENT.

THE question of the constitutional versus the local treatment of eczema has been debated for more than half a century with varying fortune; now the constitutional and now the local triumphing as the only effectual means of combating the disease. Forty years ago Dr. Hunt wrote: "It is painful to contemplate the unnecessary torments of a patient who has been tarred externally and internally or tortured with caustic for weeks together without the slightest relief, and whom the abstraction of a pint of blood and the exhibition of a smart purgative, would deliver from more than half his suffering."

Again he says: "To prevent the eruption spreading many alterative remedies have been tried with various degrees of success. It is not necessary for me to enumerate them, my whole attention having been directed for many years to one antidote alone, which, under proper regulations, acts with such uniform efficiency as to leave nothing to be desired. That remedy is arsenic."

About the same date Hebra, then Professor of Dermatology in Vienna, to whom the great advances in local treatment are mainly due, wrote: "The insufficiency of the so-called specifics in the treatment of chronic diseases of the skin, and not any spirit of opposition or desire for novelty, obliged me to desire other means of arriving at the desired end." One after the other he discusses the means of constitutional treatment—diuretics, he says, have all proved useless, cantharides injures the patient without curing his eczema, and cod-liver oil, though of the greatest service as a local application, is quite useless when given internally.

Twenty years later he said, of arsenic, that he could not concede to it the undefined blood-purifying and eczema-curing powers which were attributed to it by English and French

physicians, and therefore depended almost entirely upon local means for his undoubted success in treatment.

A very important and interesting discussion on the use of arsenic in eczema was carried on last year in America.

It must be remembered that whereas in England we have a tendency to regard many diseases or symptoms of unknown origin as possibly due to gout, so in America malaria is the cause to which are attributed similar difficulties, and therefore, bearing in mind the influence of arsenic over malaria, we must properly discount the opinions of American practitioners. One or two are as enthusiastic as was Hunt in their appreciation of arsenic, but the general tendency is to deprecate its unvarying use and general application in eczema.

The final propositions of Dr. G. H. Fox, as stated in the Journal of Cutaneous and Venereal Diseases, fairly express the views of American dermatologists of to-day on this subject, *i.e.*, firstly—"There are some forms of chronic inflammatory skin disease, and possibly some affections of a malignant type, in which the internal use of arsenic will undoubtedly exert a beneficial influence."

Secondly—"In most cases of inflammatory skin disease, regulation of the diet, and such hygienic and medicinal treatment as tends to improve the general health of the patient, will do infinitely more good than the continued administration of arsenic."

These propositions, neither optimistic with Hunt, nor pessimistic with Hebra, very fairly express the main opinion of to-day, and would appear to be supported by the observations of Murrell and Ringer, as quoted by Lauder Brunton.

They found that in frogs poisoned by arsenic the cuticle could be stripped off the whole body with the greatest readiness within a few hours after its administration.

Miss Lunn found this condition to depend on softening of the protoplasm of the columnar layer of cells in the epidermis, so that the cuticle remained attached to the dermis only by a few protoplasmic threads.

From physiological considerations, therefore, it would appear that as arsenic causes "increased metabolism in the cells of the epidermis," and therefore causes increased congestion, it would intensify itching and aggravate the symptoms generally of an acute condition.

Practically these results do follow the use of arsenic in acute eczema, and it should therefore be avoided in such a condition. Indeed it may be taken as established that arsenic does harm in acute eczema, and that it is of the greatest utility in the chronic form, where there is much infiltration of the cutis and the epidermis is thickened.

Arsenic may be given in various forms, but should always be taken during or immediately after a meal.

Arsenious acid may be given in a pill, $\frac{1}{30}$th of a grain with 1 grain of the extract of gentian, and increased to $\frac{1}{12}$th of a grain, or Fowler's solution three drops for a dose may be given twice or thrice daily.

If the remedy is doing good, the dose should be increased until as much as ten drops are taken thrice daily.

If our patients complain of colicky pains, and mucous or dysenteric diarrhœa, or if there be irritation of the eyes or running at the nose, the arsenic should be discontinued for a time.

In the sense that there is no specific constitutional treatment for skin diseases, Hebra was no doubt right, and his crusade against the indiscriminate use of arsenic has done much good, by forcing attention to each disease and its treatment on its merits; but his attitude is absurd, and his views extravagant when he speaks of constitutional treatment doing no good.

There are two constitutional conditions or diatheses, the gouty and the strumous, in which internal treatment is invaluable in conjunction with local measures, and I think the tendency of to-day would be to reverse the dictum of Hebra, and to attach all the importance to the internal, and little to the external use of cod-liver oil in strumous eczema.

The skin condition may be due in the first place to local causes, but given a healthy body it speedily gets well; given on the other hand a pale pasty child, whose glands tend to enlarge and become caseous, whose wounds show little tendency to heal, and in whom suppuration rather than resolution is the rule, what is more certain than that the eczema will spread, and, until the bodily condition is improved, will grow worse instead of better under the influence of local remedies, or at best, improve slightly, only to relapse into a deplorable condition?

If Hebra thinks more of the external than the internal use of cod-liver oil, it can only be because the oil not only exercises a beneficial local influence, but is also absorbed. In gouty people nothing is more difficult to treat than eczema—they are particularly liable to eczema of the finger tips, and of all kinds of eczema this is the most intractable, until local measures of some severity have been supplemented by the use of alkalies.

I had recently under my charge a hale but gouty publican, who had been using under medical supervision for more than a year the same remedies with which I was fortunate enough to effect a speedy cure, after assisting their action by the internal administration of iodide of potassium.

With the exception of diseases of nervous origin, such as Herpes Zoster, which tends to get well irrespective of treatment, and pemphigus, which gets well under the influence of arsenic (and even these will benefit from additional local treatment), all skin diseases require local treatment. Whatever their origin, whatever the diathetic condition, the skin itself becomes diseased, and we can influence it best by local treatment, but the most philosophic course, as well as the safest, will be to steer between the Scylla of Hunt and the Charybdis of Hebra, and combine the best of the treatment of each.

For a disease which varies so much in different individuals as eczema, it is out of the question that we should find any specific remedy.

The dry irritable eczema of the gouty drinker will not yield to the same treatment as the pustulating discharging skin of a strumous child; in each case we must observe general hygienic laws, regulate the bowels, order a suitable diet, and to our local measures add the special internal treatment the diathesis demands. Nothing is better than iodide of potassium in small doses for a gouty eczema, or cod-liver oil for the strumous form.

While there is no internal specific, some remedies, as we have seen with arsenic in the chronic form of eczema, do seem to have a special influence on the skin.

Piffard, a modern American authority, recommends the use of calx sulphurata and viola tricolor in eczema.

In eczema rubrum, especially in infants and children with

much soreness and irritation, calx sulphurata, in small doses, grain $\frac{1}{60}$th to grain $\frac{1}{20}$th, he considers often proves of great service.

"Again, in cases in the second stage, when the exudation is abundant and purulent, it should be given in larger doses, grain $\frac{1}{20}$ for children to grain $\frac{1}{5}$ for adults. Thirdly it may be used with very manifest advantage when the patches of eruptions are greatly infiltrated, and the inflammation is of a subacute character. If the effects of the drug are not evident at the end of two weeks, it will not be worth while to continue the drug longer."

The infusion and fluid extract of viola tricolor have been found of use by French and American dermatologists; it increases the amount of urine, and the total quantity of solids excreted with it; but it is rather in virtue of its depurative action that it has been found useful in eczema. It is in cases of eczema capitis in children, with formation of pustules, and tendency to become chronic, that its use is indicated. If the eruption be acute the dose should be small, if chronic or indolent the dose may be increased.

One drop of the fluid extract two or three times a day is enough for an infant, whereas for an adult it may be given in doses of from 5 to 60 drops thrice daily.

Before leaving the subject of constitutional treatment, it is necessary to refer to a procedure which has been practised by Mr. Lawson Tait and Dr. Kidd, a leading homœopath, in some cases of general eczema in young children, which had resisted a prolonged course of general treatment, namely revaccination.

Perhaps with a conscientious desire to be a consistent homœopath, Dr. Kidd recommended this course, the principle of *similia similibus* suggesting the remedy, since the disease was supposed to have originated from the first vaccination.

In all the cases recorded, three, I think, the result was most successful, and one can only wonder that so speedy and easy a cure is not more often resorted to, or at least, that successes and failures in the process have not been more often recorded.

Personally I have no practical experience of this method, and should as soon think of putting a child into a fever ward as vaccinate it in order to cure eczema.

It is well known that any general febrile disturbance, dependent upon an exanthematous cause, will for a time suppress the evidences of eczema, which will recur when the exanthem is recovered from. The re-appearance is, however, not constant, the eczema is sometimes lost with the other disease, and ceases to affect the patient. To some chance, such as this, I think it more reasonable to attribute improvements after vaccination, rather than to vaunt an isolated success as a triumph of principle. Nothing is more common than for a doctor to be asked if vaccination has not been responsible for the eczema which followed it. Never was the *post hoc propter hoc* argument more specious. Most surely eczema does often follow vaccination, but it is no more caused by it than it is by indigestion. They both, without doubt, try the strength of an ailing child, and make it ripe for the outbreak of any disease to which it may be predisposed, but to be causative, in the real sense of the word, they have no claim.

A very interesting and important question has been raised as to the propriety of healing completely a discharging eczema, and the fear of driving in the disease has often deterred practitioners from effecting a speedy cure.

Hebra and his school laugh at this idea, and no doubt in the majority of cases with good reason.

The question at issue, whether an habitual discharge may be suppressed without danger to a patient, depends, I think, upon the patient, and not on the disease.

I attended a child who had lost two young brothers from acute tuberculosis. He had a very extensive eczema of the scalp and face, but otherwise appeared in good health. Under appropriate remedies his eczema rapidly got well, but its disappearance was attended with all the signs of acute hydrocephalus, from which he soon died.

With the family disposition to this disease, it is not unlikely that the child might have succumbed to tuberculosis, had the eczema not been cured; but I fear my treatment was injudicious, as the child's disposition to disease, of a special and serious kind, was not taken into account.

Many experienced practitioners, I know, hold this view, and use the seton to keep up a slight but harmless irritation where a long-standing discharge, as from pustular eczema is

being suppressed. There is not any harm likely to be done by this, perhaps, indeed, much good, but I think it will be found sufficient if we keep the bowels acting rather more freely than is absolutely demanded by the requirements of an ordinarily healthy condition. For this purpose I combine with anti-eczematous treatment small doses of calomel, and have no reason to regret this practice.

Analogies of the impropriety of rapidly suppressing habitual discharges are common enough. Hæmorrhages from the lungs or stomach occur often enough, when hæmorrhoids, which have been bleeding for years, are suddenly cured, and I have seen cases in which cerebral hæmorrhage has seemed to follow rapid cure of an old ulcer. I am not prepared to argue a *post hoc propter hoc* relation, but it is one which appeals very forcibly to the lay mind, and we are bound to consider it; certainly the attitude of those who laugh at the notion of metastasis, because they have seen no cases, is not to be commended.

Specialists rarely see their cases from start to finish, and it is to the general practitioner and not to the specialist that we must look for a solution of this problem.

LECTURE IV.

ECZEMA: LOCAL TREATMENT.

THE question of the propriety of local treatment having been settled, we are met at starting by the two questions—What do we expect to obtain by local treatment? and by what means may we attain our end?

To the solution of both these questions, Dr. Unna, at the recent meeting, in Dublin, made very important and satisfactory efforts.

Hitherto in dermatology clinical skill has furnished the basis of practice in a spirit of the purest empiricism; but now the axiom of Hippocrates that "Law rules all things," has, to some extent at least, been supported by the recent work of Unna and others in the field of dermal therapeutics.

As was to be expected, a thorough knowledge of the structure and function of the skin is at the bottom of this development.

For the purposes of this lecture one need refer only to the horny epidermis, and the superficial vessels, ready, under the slightest irritation, to become congested, and furnish in excess materials for the manufacture of the three main products of the skin. These are:—

1. The watery vapor or insensible perspiration,
2. The fluid watery sweat,
3. The fatty sweat.

The watery vapor constitutes ⅞ths of the whole of the watery evaporation from the skin, and if suppressed, as by a hot bath, is compensated for by the increase of the fluid sweat, which can, as we shall see, be used for softening the horny layer.

The fatty secretion can be restrained by the permanent water bath, a method of treatment with which we are not familiar in England, and which has, I believe, been used in Germany only in extreme cases of burns and intractable in-

stances of Pityriasis Rubra, but the result has not been such as to justify enthusiasm for the means. On the other hand the fluid sweat can be easily restrained by means of inunction of fat, and to an even greater extent by the use of air and water-tight materials, such as gutta percha or india-rubber. Not only by these means does the subjacent skin become saturated with water, but the normal proportion of fat and water becomes disturbed, and the fat becoming relatively less, one of the main obstacles to absorption by the skin is diminished.

In the commoner skin diseases we have on the one hand, either lying under a normal or only slightly hypertrophied epidermis, a focus of disease which we desire to reach, or as in Psoriasis, an enormously increased epidermal mass which prevents the absorption of our remedies. On the other hand a great portion of the epidermis may be absent, but what remains is so swollen and distended with the products of inflammation, and the centrifugal tendency is so pronounced owing to excessive secretion, that the possibilities of absorption are reduced to a minimum. To this class belong weeping eczema and impetigo.

In the first class of cases, in which we seek to procure absorption of our drugs, and the normal or hypertrophied epidermis is the obstacle, we have recourse to measures either mechanical or chemical. By chemical means we can reduce the mass of fat-laden epidermis, and so get our remedies absorbed. McCall Anderson has popularised in England the use of caustic potash for this purpose; to Hebra we owe the similar use of sapo viridis, or potash soap; and to Unna the recent introduction of salicylic acid for the same purpose is, I think, due.

Anderson, in his treatise on eczema, and again in his recent work on skin diseases, advises the use of potash soap in the following manner:—"If the infiltration is slight, or the rash extensive, soft soap should be used. A piece of flannel, dipped in a solution of it, should be rubbed as firmly as possible over the affected parts night and morning, and the solution allowed to dry upon them; or a piece of flannel wrung out of the solution may be applied to the part, and left in contact with it all night, if the patient can bear it.

"A more elegant preparation is liquor potassæ, which may be painted over the eruption once daily with a large brush,

its irritant properties being neutralized by means of tepid water, if the smarting becomes excessive.

"Instead of soft soap or liquor potassæ, solutions of potassa fusa may be used. In the mildest cases, with only slight infiltration, two grains of potassa fusa—in the more severe, five, ten, twenty, thirty grains, or even more—in an ounce of water may be employed; but I rarely resort to a stronger solution where the eruption is extensive.

"Even the solution containing thirty grains to the ounce, which may be applied in the same way as liquor potassæ, must be used with great caution, and soon washed off with water, and the application should not be repeated oftener than once daily at the most.

"When such a strong solution is prescribed, and especially if the eruption is extensive, it is advisable for the physician to apply it himself, at first at all events; and in no case should it be used so strong, or allowed to remain on so long, as to produce any manifest destruction of the skin."

In cases of eczema sclerosum, Hebra used a solution of one drachm of potassa fusa in two drachms of water, in the following manner:—"After the accumulated masses of dead epidermis in the form of scales, crusts, etc., have been removed by appropriate means, so as to expose the subjacent red, infiltrated, moist surface, the solution is applied with a charpie-brush, passed quickly and evenly backwards and forwards over the affected part in every direction; then the hand or a piece of flannel is to be dipped in water, and with it the lotion spread more equally still over the whole surface. A white froth, not unlike soap suds, will soon be observed to form on the eczematous patch; and this only occurs when water is thus rubbed in after application of the caustic solution.

When this part of the process has been thus finished, a considerable quantity of fluid exudes in drops upon the surface. To allay the pain, and to prevent the formation of scabs, rags dipped in cold water should be applied, and frequently changed during the day.

The patient need not be disturbed during the night, if care be taken that the rags are kept wet, and this is best done by help of oil silk or gutta percha.

"After water dressing has thus been constantly applied for

a week, the minute raw places of greater or less depth, which the caustic application has here and there produced, will have recovered their epidermis, and the itching, which has quite ceased during this time, will again become troublesome. Moreover it will soon be observed that red spots re-appear here and there, and vesicles are seen; this should lead to a repetition of the former procedure. When the water-dressing has entirely removed the traces of the second caustic application, a third should follow, and this weekly course of treatment should be repeated as often as the symptoms just mentioned return. It is scarcely ever necessary for the solution of potash to be used more than twelve times, even in the most severe cases; for even if after this a few small places should show a disposition to relapse, some more gentle treatment will suffice to prevent it."

This very drastic treatment is of course only necessary in the most obstinate cases; but I have found it of the greatest service in cases of long standing dry eczema of the fingers, especially when the application has been followed by the use of a two-grain solution of acetate of lead in an ounce of lime water. This should be used on rags soaked in it, and evaporation prevented by a covering of oil silk or gutta percha tissue.

In the not unfrequent cases of callosities of the palms of the hand or soles of the feet, a milder treatment is effectual. Either lumps of sapo viridis may be put on the places and bound firmly on with bandages, or the spiritus saponis alkalinus of Hebra may be rubbed firmly in twice a day. This is prepared by putting four ounces of sapo viridis into two ounces of spiritus vini rectificati, and filtering after twenty-four hours. It should be well rubbed in with a sponge previously dipped in hot water. A good lather will be produced, and should be allowed to dry in.

I have obtained good results also in these cases by painting on the following preparation with a camel's-hair brush:

Salicylic acid, 30 parts;
Ext. cannabis Indicæ, 5 parts;
Collodion, 100 parts.

Incidentally I may mention that in a few cases of warts that have resisted all other treatment, this preparation of salicylic

acid has brought about a cure; but in others its use has been followed by failure. In other cases where the infiltration is not excessive, and the horny layer not greatly increased, Unna prefers to proceed by means of what he calls plaster mulls.

These are made by spreading the medicament employed on a sheet of gutta-percha tissue, which is incorporated with a sheet of mull, undressed muslin. Adhesion of the drug to the skin on the one hand and the mull on the other is secured by the addition of some substance, such as the purest india-rubber or purified oleate of aluminium. These are both indifferent to the skin, and cause the plaster, drug and skin to stick together. By this means a large amount of the drug employed is brought into direct contact with the skin, while its absorption is facilitated by means of the impermeable covering. The watery sweat cannot escape, and saturates the diseased part, while the fatty sweat which forms the chief obstacle to absorption becomes relatively less, and absorption takes place freely and easily.

By these means the first difficulty of procuring absorption of drugs is overcome, and these methods are of the greatest value in all chronic cases such as eczema squamosum, lupus or indurated acne. The glycerine gelatines are of use in cases of acute eczema, erythema or diffused eczema with much discharge, especially when the horny layer is wanting and its fatty matter deficient. Here our object is to procure the absorption of the secretions, and dry up the mass while the epidermis has time to be reformed. For this purpose rice powder may be used, but the gelatine compounds are especially serviceable. They permit the escape of the insensible perspiration, so no objectionable results to the lungs or kidneys occur. They cause no distress to the patient, and protect the affected parts from exposure to the air and contact with injurious substances. They are prepared by mixing gelatine and glycerine in definite proportions with the addition of the medicament desired.

A sufficient quantity of the compound is melted in a water bath or in a spoon, and painted on the part affected with a brush. It soon dries, and may then be covered with a bandage where friction by the clothes may disturb, but on the face it need not be covered; and as it is barely noticeable, will be of

inestimable value to people who have facial eczema, and still may have to be engaged in business. According to Unna, the glycerine gelatines are of the greatest utility as auxiliaries to other dressings.

Besides effecting the cure of inflamed surfaces, they ensure rest, immovability, and, if covered by bandages, an increased compression. In the case of ulcers of the leg, it is of great advantage to paint the healthy skin thickly round the sore with zinc gelatine, and then to bind the whole of the leg firmly with a double-headed bandage. Under this bandage the accompanying eczema heals, the varicose veins improve, and the patient cannot possibly do himself any harm by scratching.

For the better class of patients I feel sure these methods will in time be universally adopted, but motives of time and expense will prevent their universal adoption amongst our poorer patients, and for these we must consider how to use the lotions, powders, and ointments of our pharmacopœia.

In the choice of remedies we are guided firstly by the stage of the disease, by its acuteness or chronicity, and secondly by its situation. If acute, our object is to soothe and allay irritation, to prevent, if possible, exudation, and so check the further spread of the disease. For these purposes we have to apply sedative drugs, and above all to exclude all external irritants. Of these the commonest are air and water. If the stratum corneum be absent, the soft and succulent cells of the rete malpighii rapidly absorb water when in contact with it, and so become swollen and burst, with the production of increased redness, swelling, and irritation.

It is always advisable for a patient with eczema in the erythematous or vesicular stage to abstain altogether from the use of water. If, however, its use be insisted on despite advice, it can be rendered less harmful by the addition of a little glycerine or common salt.

More harmful than water alone is soap and water. To say nothing of the inevitable friction, soap is prejudicial from its composition. It always contains an excess of free alkali, whether soda or potash, and therefore acts as an irritant. Moreover, it combines with and removes the oily matter of the skin, which is rendered dry and harsh. For this reason ordinary soap is most objectionable in eczema, and indeed its use ought to be interdicted amongst all who have a tendency

thereto. Eczematous patients, even when well, commonly have dry rough skins; and a very little extra dryness, such as that caused by exposure to wind or over much washing, often starts a fresh outbreak of the disease.

In my experience medicated soaps do very little more good than those in common use—they all do harm on account of their free alkali depriving the skin of its oily protection. To meet this difficulty, Unna suggested what he calls an over fatty soap, which contains four per cent. of unsaponified oil.

Mr. Perry, chemist, of the Hagley Road, has made a similar soap for me, which will, I think, prove of great value to people predisposed to eczema, and to those whose skins are dry and harsh owing to a deficiency of fat. This soap will minimise the evils of washing in cases of eczema, and to that extent will be useful; but it is rather in the prevention than in the cure of eczema that its very great value lies. Though we owe much to Unna for his other preparations, this soap is, I believe, destined to prove the most useful of them all.

The glycerine gelatines, especially those containing lead or zinc, best serve our purpose in treating acute eczema, for they offer a protection against external irritants, and at the same time encourage absorption of the watery products of inflammation, while the fatty ones are stored up and help in reforming the thinned or lost epidermis. Though very little absorption of drugs is possible when used in this combination, very little is needed—we have only to allay the symptoms, to diminish congestion if the disease be only erythematous, and to dry the skin if the eczema be of the weeping variety.

We may gain our ends also by the use of powders and lotions, especially such lotions as deposit powders when drying up.

Of powders, simple rice powder, powdered starch or talc, answer very well, whether used alone or in combination with zinc oxide or oleate, which exercises a slightly astringent action. To these camphor may conveniently be added, if there be much itching; and a useful formula is the following:

℞—Zinci oxidi.
Pulveris amyli āā ℥ ss
Camphorae gr. 20.
M. S. to be used as a dusting powder.

Care must be taken, in using powders, not to allow them

to cake on the skin or become worked into a paste, especially in the intertrigo of young children.

Lotions should be applied on rags soaked in them. These should not be too thick, and should never be covered with gutta-percha tissue to prevent evaporation, for they do good partly by evaporation and partly by the powder they leave on the skin.

To promote this end they should contain a little glycerine, which, as Bulkeley points out, facilitates the adhering of the powder, and keeps the parts supple.

Some lotions are particularly valuable in the first and second stages, when the vesicles are just rupturing, and the irritation and itching are at their height. One of the most valuable of these is lotio nigra, which must of course be shaken before being used.

Among other useful combinations are the following:

℞—Plumbi acetatis
Zinci sulphatis, āā gr. 40.
Liq. carbonis detergentis, ʒ ij.
Glycerini, ℥ j.
Aquam calcis ad. ℥ xx.
M. ft. lotio.

℞—Acidi boracici, ʒ v.
Acidi hydrocyanici dil. ʒ ij.
Glycerini, ℥ j.
Aquam ad. ℥ xx.
M. ft. lotio.

It is easy to multiply prescriptions for lotions, but as a matter of practice one gets into the habit of using only a few remedies, and indeed the most important part of successful dermal therapeutics lies not in the number of remedies which one can employ, but in a recognition of the right time to use them—when, that is, to encourage or check transudation through the skin by the use of lotions or ointments; and at the risk of wearisome iteration it is necessary again to emphasise the necessity of using lotions or powders in the acute conditions, and ointments in the chronic.

With respect to the above-mentioned lotions, it is important to bear in mind that some skins tolerate tar preparations very badly, whereas others are irritated by the use of glycerine.

To some extent, then, the use of our lotion is experimental, and it is wise to begin with small quantities of the liquor carbonis detergentis, which is a patent preparation of Wright's, and generally very much relieves itching, but sometimes causes a good deal of burning pain.

When the disease is in the stage of pustulation, and a good deal of pus is exuded, and scabs are freely formed, oily preparations are of most use. Here again the tarry oils must be used with great discretion, as though invaluable they are apt to inflame some skins if used in too great strength. It is well to combine them with cod liver oil, or castor oil, or olive oil, as in the following prescriptions:

℞—Olei cadini, ʒ iv.
Olei morrhuae ad. ℥ iv.
M. ft. lotio.

℞—Olei rusci, ʒ iv.
Olei ricini, ℥ j.
Olei olivæ, ℥ j.
M. ft. lotio.

A patient preparation of this kind of is the so-called linimentum plumbi lactatis co, which perhaps is the best of the oily remedies in use.

A drug I have been using lately with the greatest advantage is the Oleum Deelinæ. This owes its name to the Dee Oil Company, who prepare it. I prescribe it in the following combination, varying the strength of the prescription as the patient becomes tolerant of the oil.

℞—Olei deelinæ, ʒ j.
Olei ricini, ʒ ij.
Olei olivæ, ʒ iij.
M. ft. lotio.

It is a little known preparation, but invaluable. In common with all oily preparations, it is used by spreading lint soaked in it on the diseased part. Lotions can be used easily in the daytime; but, unless they are constantly renewed, the lint saturated with them soon becomes dry. For this reason they are difficult to use during the night, and it is then that the oily preparation may be used with advantage.

Ointments may be used towards the end of the second

stage of eczema, when the very profuse serous discharge has ceased and there is a tendency to the formation of crust, and some slight exudation into the skin; but they are especially valuable in the chronic form of the disease when the skin is thickened and leathery. They are of use in proportion to their power of excluding irritants, and to the ease with which they are absorbed. Both of these ends, as mentioned before, are best served by the use of Dr Unna's plaster mulls, but we may to some extent gain the same end by covering lint, spread with ointment, with gutta-percha tissue.

The number of ointments is legion, but as a matter of fact no practitioner uses more than two or three. Most others use zinc ointment, or, preferentially, that made with the oleate of zinc.

If there be much itching, it is wise to add half-a-drachm of carbolic acid to each ounce of this. If the case be obstinate, and if there be no long-standing venous engorgement as from varicose veins, a useful preparation can be made by mixing the zinc ointment with the ung. picis liquidæ of the Pharmacopœia in the proportion of 6 to 1. If this amount of pitch be found to be tolerated, the quantity may be increased; but it is wise to be careful with it, especially in elderly people. Young plump skins seem to tolerate tar much better than the old and shrivelled.

Of mercurial ointments, that made with the oleate is I think best. It is commonly made of the strength of 5 or 10 per cent., and should of course be used with discretion. It will be found a very valuable adjunct to the internal treatment of syphilitic sores, especially if a little morphia be added to the ointment.

The difference this addition makes is almost incredible; like many good things it comes to us with the sanction of long years, for it was freely and successfully used in Naples before the days of mercury. Chrysophanic acid ointment must be carefully avoided in acute eczema—it does little if any good in the chronic form, and is occasionally very prejudicial if too freely used on the head.

The influence of the localization of the disease on its management is sometimes considerable. This is especially true in eczema of the scalp, owing to the sticky exudation matting the hair together, and to the free discharge of pus. It is im-

possible to get to the seat of disease without removing the hair. This may be best done by clipping it close to the scalp. Very frequently, in hospital practice, these cases are seen to be due to the presence of pediculi, which must, in the first instance, be got rid of. This can be done by washing the head with equal parts of petroleum and olive oil. Then the crust must be removed, but often at the expense of much time and trouble.

The head should be saturated with olive oil, and after a few hours the scalp should be washed with soap and warm water. This will probably get rid of the crusts, and then oleate of zinc ointment should be freely rubbed on the surface.

The ointment should be renewed once or twice daily, and crusts or old ointment removed whenever discovered.

This form of eczema commonly occurs in delicate or strumous children, and the internal use of cod-liver oil is of the first importance in the treatment. They soon do well if, after the first washing, no more water be used, and the ointment be thickly applied.

When the discharge ceases, and the scalp remains dry and scaly, it is well to add a little unguentum picis liquidæ to the unguentum zinci, or better still to use the oleum deelinæ, in the proportions mentioned before.

For a similar dry eczema of the scalp, in which some irritation is associated with excessive formation of scurf, Piffard recommended the use of the following preparation—

℞—Acidi salicylici, grains x to xx.
Ol Lavandulæ, ʒ iijss
Ol Citronellæ, ʒ ss
Ol Pini Sylvestris, ℥ ij
Ol Ricini, ℥ jss
℥

In this preparation the salicylic acid is designed to restrict the formation of scales, the oil of the pinus sylvestris acts as a stimulant, and the castor oil to correct the drying effects of the latter. The best way to apply the oil is from a small oil can. The hairs having been separated a few drops are applied directly to the scalp, and gently rubbed in with the finger. The application must be made daily at first, but afterwards at longer intervals.

Eczema of the hairy portion of the face may be treated like eczema of the scalp, provided there be no formation of pustules and infiltration of the skin. In the latter cases every hair on the affected part must be pulled out. As a rule they come out easily, as their attachments to the follicles have been loosed by inflammation. Zinc ointment must be rubbed on, and, according to Piffard, calx sulphurata, in doses of grain $\frac{1}{10}$th to grain $\frac{1}{5}$th administered twice daily.

Eczema of the scrotum is characterised by great itching and extensive infiltration, and its treatment is most difficult, owing chiefly to the dependent position of the part. The scrotum should be supported by a suspensory bandage, the bag being made of, or lined with, india-rubber. This, of itself, often causes marked improvement, but if our patient will permit it, it is next advisable to scarify the scrotum and let blood flow freely. The patient should sit in a tepid bath to encourage bleeding, and after drying the parts a little tincture of benzoin should be applied. The scarification will have to be repeated in a week or two. If this procedure is objected to, a solution of liquor potassæ must be applied as before directed, and after the irritation has subsided, zinc ointment is to be rubbed freely on.

Eczema of the legs, if chronic, is best treated by the use of Unna's impermeable dressings, but if associated, as so frequently happens, with varicose veins, may be cured by applying zinc ointment spread on lint, and covered with one of Martin's bandages. If the condition be sub-acute it is best to apply linen soaked in boracic acid, and covered with gutta percha tissue, or sometimes merely rags, saturated with the linimentum plumbi lactatis co.

Of course a very important part in the treatment of these cases consists in the maintenance of the horizontal position. It is best to keep the patient in bed, and to get rid of the weight of the bed clothes by means of a cradle.

Eczema intertrigo, which so often occurs in fat young children or obese adults in the fold of the groin or beneath pendulous breasts, should be treated by dusting on oleate of zinc and starch in equal parts, with the addition of a little tannin, if there be much secretion.

Eczema of the nipple has assumed much importance since the publication of Sir James Paget's paper on the subject in

the St. Bartholomew's Hospital Reports for 1874. He drew attention to "a disease of the mammary areola preceding cancer of the mammary gland." He described the condition of the skin as resembling chronic eczema in certain features, with an intensely red raw surface, very finely granular, giving rise to a copious, clear, yellowish, viscid exudation; the sensations were commonly tingling, itching, and burning.

In some of the cases the eruption presented the character of an ordinary chronic eczema, with minute vesications, succeeded by soft moist, yellowish scabs or scales, and a constant viscid exudation. In some it was like psoriasis, dry, and with a few white scales slowly desquamating. In both these forms, says Paget, I have seen the eruption spreading far beyond the areola in widening circles, or with scattered blotches of redness covering nearly the whole breast. This is not a true eczema, but probably a slowly advancing cancerous change near the mouth of the lactiferous ducts, which at a very early stage leads to irritative effects on the superficial tissues of the nipples and surrounding skin, and eventually penetrates into the substance of the mammary gland. The diagnosis is difficult, but if the disease does not yield soon, as would an ordinary eczema, to general treatment, its true nature should be suspected.

Eczema is very often set up by and associated with scabies, and must be specially treated after the acari have been destroyed, but as will be seen later, ought not to be confounded with that disease. Generally speaking the modern and wide definition of its name renders its diagnosis easy, nor with the exception of a few cases of squamous eczema ought there to be any difficulty in its recognition. I have drawn largely on the literature of the subject, but am especially indebted to the writings of Unna, Piffard, Duhring, Bulkley, Hebra and McCall Anderson.

LECTURE V.

PSORIASIS.

NEXT to eczema and scabies Psoriasis is the most common of skin diseases. The frequency of its occurrence renders its diagnosis a matter of importance, while the difficulty of deciding, in exceptional cases, whether we have to deal with eczema squamosum, a squamous syphide or simple psoriasis makes it necessary to carefully review its pathology, its ordinary situation and clinical characteristics. A simple case cannot be mistaken for anything else, but every now and then we meet with cases which require the utmost care in diagnosis. It may be defined as a disease of the skin which is chronic, usually relapses, is symmetrical in its localizations, and is associated with the formation of profuse quantities of silver grey scales, which can be removed readily by the finger nails, upon more or less large reddened parts of the skin, which are elevated to a trifling extent.

The pathological element in psoriasis is great enlargement of the papillæ, with marked distension of the blood vessels of the skin. Neumann found the papillæ elongated twelve to fifteen times the normal, their tissue infiltrated with round cells, the coils of vessels dilated in some places, narrowed in others; in the upper parts of the cutis the bundles of connective tissue are broader than normal, and the dilated vessels surrounded by proliferated round cells. Upon the areas of enlarged papillæ are formed in excess the epidermal scales which give to psoriasis its clinical characters.

At first the collections of scale are discrete and punctiform. They soon increase in size, and constitute the common form of the disease known as psoriasis guttata, in which the body is more or less covered with white raised scaly spots more or less widely separated from each other by healthy skin. These increase at the periphery and form masses of varying

size often running into each other, and, according to their arrangement, giving names to different varieties of the disease. These varieties do not, as in the case of eczema, vary in their pathological anatomy, and there is no object in specially naming them. Locality has very little influence in modifying the appearances; only when the disease affects the scalp and nails does psoriasis require special description.

On the scalp it occurs in irregular streaks of eruption, very closely resembles seborrhœa sicca, and, if unaccompanied by general psoriasis, is difficult to diagnose. There is no discharge as in eczema, nor are the hairs so much matted together.

The nails are thickened at the free edges, of a dark yellow or brown color, lustreless, laminated and brittle. They often break away in patches, leaving portions of the matrix uncovered.

The disease commonly breaks out simultaneously at several points, but almost invariably the backs of the elbows and fronts of the knees are the first parts to suffer. It may occur anywhere, but chooses especially parts where there has been pressure, as from garters or a belt. Patches of psoriasis tend to disappear, leaving only a pigmented surface, but unless treatment is adopted and persevered with, the body is rarely altogether free from the eruption. It is not only chronic but relapsing, and, like many other skin diseases, has a tendency to reappear every spring and autumn.

If it occur on the face, its unsightliness is a sufficient reason for treatment, but otherwise, as no symptoms beyond more or less itching are complained of, it will often be found to have existed for years before it is brought under medical notice.

No organic disease or visceral disturbance is constantly associated with it, and indeed those who suffer from psoriasis commonly enjoy robust and vigorous health. As to its causation, nothing definite is known; it occurs among the rich equally with the poor. It is associated with no diathetic disturbance, and beyond a strong probability that it is hereditary its etiology is a blank. Recently it has been added to the victims of the all-devouring bacterial pathology, but as yet neither has its course been modified, nor its treament altered thereby.

In determining whether a rash is simple psoriasis or a squamous syphide, there is, as a rule, no difficulty. The history of the patient, the position of the rash, concomitant signs of syphilis, or their absence, render the diagnosis easy.

Sometimes, however, we are met by cases which almost baffle correct diagnosis, and in view of such, it is important to have a clear idea of the difference between the two diseases.

Fournier, the great French Syphilographer, has drawn up the following scheme of distinction:—

1. *The Volume.*—In papulo-squamous syphilide the eruption is characterized by papules, generally small or of medium size, while in psoriasis they are large, unless in psoriasis guttata, and frequently form extended plaques.

2. *Color.*—In syphilis the papules are of a dark red, often of a characteristic raw ham color. In psoriasis they are rose colored, or at least of a lighter red. Still both diseases have often quite a similar color.

3. *Desquamation.*—Here we have a marked difference. Syphilitic papules are slightly and only partially squamous, often being free from scales over a large portion of their surface, and the scales are thin, small, superficial and grey. In psoriasis, on the contrary, they are abundant, the scales cover the whole or greater part of the papule or psoriatic patch; they are large, thick, heaped-up, stratified, white and form a laminated covering, often a true epidermic carapace.

4. *Results of Scraping.*—In a papulo-squamous syphilide the papules do not show, upon scraping with the finger nail, the micaceous striac and the candle grease drop (*tache de bougie*) which is characteristic of psoriasis, and pathognomonic when well marked.

5. *Consistence.*—The hardness of the papule is what I call the sign of the blind. In syphilis the papule is resistant to the touch, hard, and gives the sensation of a foreign body inserted beneath the epidermis, and recalls the parchment induration of the chancre. In psoriasis we find the skin simply thickened, but without hardness or true resistance.

6. *Localisation.*—In syphilis there is no characteristic localisation, no point of predilection, except for the palmar and plantar varieties. In psoriasis we find, in the great majority of cases, the curious localisation on the knees and elbows, which is truly characteristic.

7. *Treatment.*—While syphilis is in general rapidly modified by mercury, psoriasis is no wise affected by its use.

8. *Duration.*—In a large number of cases the duration of the eruption is an excellent sign. In syphilis it is short in comparison to the habitual persistence and chronicity of psoriasis, and scarcely ever exceeds a few week or months; while in psoriasis it remains for months and even years; the eruption is always chronic, and this chronicity is often of itself a characteristic sign.

9. *Antecedents.*—In the syphilitic contamination can usually be found, if carefully sought for, traces of the early lesions being generally discoverable; often the papulo-squamous lesions coincide with other secondary manifestations, such as mucous patches, alopecia, etc. Nothing of this kind exists in the psoriatic, unless the two diseases be present at the same time.

The treatment of psoriasis differs so widely from that of eczema that a diagnosis must be made with as much certainty as possible before treatment is commenced. As is the case with syphilis, no difficulty as a rule occurs in deciding the disease we have to deal with, but occasionally also it is next to impossible to make up one's mind. Patient's statements are so unreliable that but little value can be attached to them: were it otherwise, the fact that a chronic eczema has nearly always at some time or other been associated with a moist exudation would suffice to establish a diagnosis; for psoriasis is always and completely dry. It is always chronic and relapses with almost mathematical precision and certainty; eczema is often acute or sub-acute, and when chronic, has been acute as a rule, at the beginning; psoriasis affects the thicker parts of the skin and the most exposed regions, such as the knees and elbows; psoriasis affects the extensor aspects of joints, eczema the flexor aspects; psoriasis the parts which sweat but little, eczema those in which perspiration is common: psoriasis has always the same aspect, eczema is protean in its appearances.

The difficulty of distinguishing between these two diseases arises most frequently when we have to deal with a squamous affection of the palms and soles. This may be due also to syphilis, and while a correct diagnosis is important, it is also most difficult. Of course it is easy enough when there are other evidences of one of these diseases, but if the palmar

eruption stand alone, we shall be right probably six times out of ten if we treat the case as eczema, three times if we regard it as syphilis, and only once if we call it psoriasis.

Treatment.—When once we have made up our minds that we have a case of simple psoriasis to deal with, the treatment is easy enough. In the first place we must get rid of the scales, for no local application can do any good when applied on the top of a mass of dead epidermis. Our patients therefore should be directed to take a hot bath, and scrub themselves well with soft soap and a good stiff nail brush. When dry, the ointment considered most suitable should be well rubbed in. The local applications which, up to recent times, have been considered of most service in psoriasis are the tarry preparations; either the unguentum picis liquidum, or oleum cadini, or oleum Rusei.

Balmanno Squire some few years back suggested the use of chrysophanic acid, or chrysarobin as it is now more properly called. This is the active ingredient of Goa powder, and is used by Indians and Brazilians in the treatment of skin diseases.

The Indian mode of using the drug is to cut a lime fruit, dip it in the powder, and dab it on the affected part. The Brazilians mix it with vinegar, and so apply it, or use it as an ointment. In the latter form we find it most convenient for use, and I commonly make the ointment of the strength of ten grains of chrysophanic acid to an ounce of vaseline. Some use it in much greater strength, a drachm to the ounce, but I think it unadvisable, as, especially in recent cases, some skins are very intolerant of the action of chrysarobin.

If the disease be limited in extent, no local treatment can be so good as the use of Unna's plaster mulls, containing from 18 to 45 per cent. of chrysarobin. By their means free absorption takes place, and we can overcome the great objection to the use of chrysophanic acid, namely, the inevitable discoloration of linen and underclothing. These plaster mulls are expensive, and Messrs. Southall, of Birmingham, have made an effort to provide an efficient substitute. They have made a mixture of the drug, gelatine, and glycerine, and water in definite proportions, forming a mass which can be dissolved by gentle heat, and applied by means of a gum brush. When applied it soon dries, and whilst drying should be covered with cotton

wool or charpie, which forms a tissue containing the drug and adhering to the skin, whilst the clothes are, to a great extent at least, spared from staining.

The oil of cade should be used in cases of psoriasis of the hairy scalp, and rubbed in once or twice daily; but for other forms of the disease, except in my experience in one case only, the tarry preparations are not comparable in their good effects to chrysophanic or pyrogallic acid. They are unpleasant to use, and not so efficacious. If the bases of the eruptions are very red, irritable, and itch a great deal, we had better discard the chrysophanic for pyrogallic acid. Practically this means use pyrogallic acid in acute cases, since the older forms of the disease do not itch much.

Pyrogallic acid may be made into a good application by mixing with vaseline or lanoline, 20 or 40 grains to the ounce being used; but it should be employed with caution and its effects carefully watched, as sometimes its constitutional effects are very alarming. It should therefore be used only over a small area, and its effects watched. In nearly every case, however, we may safely use chrysarobin.

The most important part of treatment is the internal administration of arsenic. Its use alone, with the aid of soap and water, will get rid of the disease for a time, though it is better to combine it with local treatment; but even if we continue using arsenic after the disease has apparently been cured, in nearly every case a relapse is certain to occur.

A patient should be warned of this liability to relapse, and encouraged to persevere time after time with the same treatment. We very frequently see cases of more or less recent psoriasis, and very seldom cases of the inveterate type, so that it is probable that the tendency to reappearance may die out. One or two cases have certainly justified the recommendation to continue taking arsenic in small doses for a year or more.

Fowler's solution may be given in doses of from 3 to 10 minims thrice daily, beginning with the smaller dose and gradually increasing to the larger. If symptoms of arsenical poisoning come on, the use of the drug must be discontinued for a time.

The Asiatic pills of Hebra are very useful where arsenic has to be taken for a lengthy period. They are prepared by

mixing 66 grains of arsenious acid and 9 drachms of powdered black pepper with gum arabic and water, so as to make 800 pills. Each pill contains about 1-12th of a grain of arsenious acid. In most instances Hebra used to give the three pills once a day, just before dinner; but sometimes, for months at a time, he gave as many as twelve a day. We can most surely do good with arsenic internally, and chrysarobic or pyrogallic acid externally, but must never fail to remind our patients of the almost inevitable occurrence of relapses.

LECTURE VI.

SCABIES.

CHIEF among the parasitic diseases to which the skin is liable is Scabies, the Itch.

It is dependent upon the presence of the itch insect, the acarus scabiei, and is manifested partly by the effect of the acarus itself on the skin, and partly by the effects of the scratching caused by almost intolerable itching. It is only during the last fifty years that the acarus has been identified and recognized in England as the cause of this disease, though as far back as 1687 Bononio published an accurate but long-forgotten account of the acarus, which, for twenty years before Renucci demonstrated it conclusively to the savants of Paris in 1834, was the cause of keen controversy in that city.

A M. Galès, apothecary to the Hospital St. Louis, was successful in winning a prize offered to the discoverer of the itch mite, and though none of his contemporaries were equally fortunate, it was nearly twenty years before Raspail proved the itch mites of M. Galès to be cheese mites, and his discovery a fraud.

However, it is now an established fact that scabies is due to the presence of the acarus.

The male is very small, and does not burrow under the epidermis; its sole function seems to be to impregnate the female.

The latter dives at an acute angle under the epidermis, into the soft cells of the rete, through which it tunnels a passage, called a cuniculus, and as it travels deposits an egg daily. These burrows average one-fourth of an inch in length, and are indicated by the presence of a vesicle or pustule at the head of a chain of little black dots, placed at regular intervals.

The vesicle is due to the irritation caused by the acarus making its way through the epidermis, and the black dots are the eggs.

When the process of ovulation is complete the acarus hollows out a little chamber beneath the epidermis, in which it dies.

From the ova, if undisturbed, are developed fresh acari, which pass from the skin broken by friction, and keep up on the same person, or develope in others in close contact with the original sufferer, a fresh outbreak of scabies.

To a diagnosis of this disease the discovery of these burrows is an absolute necessity. An expert will be almost sure to find the acarus itself or some of the ova, but, failing this, the presence of even one cuniculus is sufficient.

The most common seat of the cuniculi is the tender skin between the fingers, and over the ulnar prominence at the wrist; but they may be found beneath the mammæ, in the axilla, or on the penis, and in children frequently on the buttocks or between the toes.

It is common to find along with the cuniculi acuminated and more or less isolated vesicles and pustules, with irregular dots or lines in the roofs.

When the nature of this disease has once been suspected, and the burrows sought for, it will rarely be misunderstood, but it is not uncommon to find the victim of scabies treated solely for eczema. Some degree of the latter is almost invariably present, as the itching, especially at night, is intolerable, and the temptation to scratch irresistible. Consequently, we find excoriation, vesicles and pustules and other evidences of an inflamed and irritated skin.

It is possible to mistake scabies for Cheiro-Pompholix (Hutchinson), in which we get vesicles between the fingers, and very distressing itching, but the vesicles soon become blebs, and if the burrows are sought for no mistake ought to be made.

Treatment.—The disease being due to a definite cause will be cured by the removal of the cause, and therefore we have first to get rid of the acari. Sulphur, iodide of potassium, petroleum, balsam of Peru, and other substances will kill the acari, provided they are brought into contact with the drug.

It is advisable to scrub the patient thoroughly well with soap and water in order to break open the burrows, and then to rub freely on the skin the ointment preferred. The patient should then go to bed, clothed in flannel garments, and next morning repeat the bath. A repetition of both inunction and

bath should be made during the next twenty-four hours, and then, beyond the use of a soothing lotion to relieve any accompanying eczema, no treatment should be employed for a day or so, until the result has been watched.

It is important to discriminate between the itching due to the presence of the acarus, and such as may be caused by eczema. The latter will be increased by undue persistence in the treatment by means of parasiticides, while if the itching is due to the acarus it will be cured.

If the burrows are broken up and the ointment well rubbed in, it will rarely be necessary to repeat this method, but occasionally some cases resist the ordinary treatment, and in such a one, after many other doctors as well as myself had failed in giving relief by ordinary means, I was fortunate enough to effect a cure by the use of Naphthol in the following combination:—

℞—Naphthol, 15
Lard, 100
Green soap, 50
Prepared chalk, 10
} M ft. unguentum (Kaposi)

For ordinary cases we rely upon the Unguentum Sulphuris of the British Pharmacopœia, or prescribe one of the following combinations:

℞—Potassii Iodidi ʒ j
Sulphuris ℥ j
M ft. unguentum

℞—Sulphuris sublimati ℥ iij.
Olei cadini
Saponis viridis
Adipis ad ℥ viij
Cretae ℥ ij
(Hebra) M ft. unguentum

A sulphur vapor bath may be used, but is not a pleasant remedy, and if the ointment be well applied, is unnecessary.

The disease is very contagious, being transmitted mainly by contact of warm bodies, and therefore patients should be isolated, while, as a matter of precaution, their clothing should be destroyed or thoroughly disinfected by heat.

LECTURE VII.

ACNE.

No skin disease is more annoying to the patient, or troublesome to the doctor. It is annoying because it occurs commonly at an age when people are peculiarly sensitive to appearance, and always on the face. It is troublesome because it is so very difficult to cure.

There are two common varieties of acne—Acne Simplex and Acne Rosacea. By the first we mean an inflammatory process (generally chronic) of the sebaceous glands, with the formation of nodules and tubercles, in the centre of which is a black comedo. Acne Rosacea is due to dilatation of the deeper plexus of blood vessels of the skin, with or without affection of the sebaceous follicles, and therefore is improperly named acne. It is manifested by erythema of the skin of the nose, cheek, chin, and forehead, and may present visibly dilated blood vessels and evidences of acne simplex, which it resembles in the chronicity of its duration.

Acne Simplex depends upon deficient activity of the sebaceous glands, which produce an imperfect kind of sebum, with a tendency to dryness in the efferent duct. From this results a varying number of black points on the surface of the skin, especially on the nose and forehead. These consist of comedones or plugs of sebum, the end of which get blackened by extraneous dirt. Nearly every one, at some time in his youth, presents evidences of this defective sebum formation, but other conditions are necessary for the development of acne. For this it is necessary that inflammation should be excited in the connective tissue round the follicles and their ducts. If the sebum merely continue to increase in quantity without inflammation, we get little round whitish nodules on the surface of the skin, as well as the black dots, but if inflammation occurs, we get first a reddened condition of the nodules and surrounding skin, and nearly always in some degree the for-

mation of pustules. These suppurate, evacuate their contents, and lead to more or less cicatrisation; but if the follicles alone be inflamed, cicatrisation is avoided. There may be hundreds of these comedones, nodules, and pustules scattered over the surface of the skin, not only of the face but also of the shoulders, which have a peculiar greasy feel.

Digestive disturbances seem occasionally to induce acne, and I have several times found it in young girls who have eaten too much meat and taken too little exercise. Anæmia also seems sometimes to be a predisposing cause, but the advent of puberty in the great majority of cases furnishes the occasion for the outbreak of the disease

What is the exact value of the generative function in the production of acne is doubtful, but the fact of its rare occurrence in eunuchs seems to indicate some connection. The establishment of the menstrual function generally determines its appearance in young girls, for while it is common in males, it occurs with greater frequency in young females, and I have known cases of adult females in whom every menstrual period has coincided with a fresh outbreak.

Certain drugs develope acne, especially tar, chrysarobin, and the salts of iodine and bromine. Tar and chrysarobin exert a local influence, whereas the excretion of the iodine and bromine salts by the sebaceous glands is the cause of their irritant qualities. The iodide eruption coincides with catarrh, and manifests itself commonly about the face; pustulation soon occurs and leaves no scar. Bromide acne occurs usually on hairy parts, especially the scalp, and, as with the iodic form, disappears or increases as the drug is withheld or increased. The treatment of these drug eruptions is therefore obvious; discontinue the drug and the acne will disappear. If, however, as often happens, iodide and bromide of potassium or sodium must be taken, we may minimise their acne-producing effects by the addition to each dose of from three to six minims of liq. arsenicalis.

As acne is chronic in its course, and tends to recur when apparently cured, its treatment resolves itself into two considerations—firstly, how to get rid of the present evil; and secondly, how to prevent relapses. The disease should always be promptly treated, owing to the desirability of preventing the formation of cicatrices. It is therefore important to com-

mence treatment before there is much inflammation. Unfortunately, we are not often consulted before this stage is developed, and we are therefore driven to minimise the evil, where some permanent marking is inevitable. As there is usually an excess of fat on the face, it is advisable to wash with Hebra's spiritus saponis alkalinus, which dries the face. For a mild case in which there are not many pustules or tubercles, I am in the habit of prescribing a sedative lotion to relieve accompanying irritation, and generally use the following lotion:—

℞—Acid boracici, ʒ iij.
Aquam ad., ℥ xij. m. ft. lotio.

Before going to bed the patient is recommended to steam the face over a jug of hot water, and then to rub the skin thoroughly with a hair glove, and lastly to sponge it freely with a lotion containing sulphur, *e. g.*—

℞—Sulphuris præcipitati,
Glycerine, ā ā ʒ vj.
Spir. vini. rectificati, ℥ iv. m. ft. lotio.

The spirit evaporates, and the glycerine causes the powder to adhere to the skin. This remains during the night, and next morning should be washed off with hot water and free sponging with the boracic lotion repeated. Instead of a sulphur lotion, the ung. sulphuris iodidi of the B. P. is sometimes prescribed, but I prefer the lotion; it is easily applied and more cleanly. If tubercles be present, Unna's salicylic or icthyol plaster should be put on each, and kept on as long as possible; if the nodules are not pustular, icthyol gelatine should be melted and painted on in the usual manner. If there be pustules, their contents should be evacuated with a needle. In addition, every black spot should be got rid of by pressing over it a watch-key. A little white plug of sebum will be expressed. This is by the laity commonly thought to be a grub, but microscopical examination proves its real nature. A parasite, the "demodex folliculorum," has occasionally been described, but is rarely to be found in these collections of sebum. Such measures will usually get rid of the disease, but it is, as we have seen, very apt to recur, and it is therefore advisable to recommend our patients to steam their faces, scrub them well

with the hair glove, wash with alkaline soap and water, and above all spend a little time daily in expressing the contents of the blocked follicles.

Failures of health must be attended to, the bowels must be kept regular, and a suitable diet enjoined; but the treatment must be mainly local, and if conscientiously carried out by the patient, will usually prove successful.

LECTURE VIII.

RINGWORM.

I AM sorry that the time at my disposal does not permit me to give an exhaustive description of the varieties of ringworm, so I purpose very briefly to review its treatment, and show how its well-known difficulty depends on the anatomical peculiarities of the disease.

Ringworm is known as Herpes Circinatus or Tinea Circinata if occurring on the surface of the body, as Tinea Tonsurans, or Tinea Tondens if occurring on the scalp, and Tinea Sycosis if attacking the hairy portion of the face. In every case it is due to the presence of the Tricophyton, a vegetable parasite, and is extremely contagious. When it occurs on the body it begins as a small reddish spot, which extends peripherally, and tends to become circular. The advancing edges may be in the form of minute vesicles, or the inflammation of the skin may evidence itself in desquamation.

The patches of course vary in size, sometimes being as large as the palm of the hand, and have a reddish surface covered with thin greyish scales, especially about the advancing edge, while the centre is paler in color and only slightly scaly. This difference between the advancing edge and the portion of skin which is earliest attacked is most marked in the rare cases of so-called Eczema Marginatum, which is in reality due to the presence of the Tricophyton.

The fungus finds its way into the epidermis, and sets up slight inflammation with either the formation of vesicles or of desquamation. If some of the epidermis be scraped off and soaked in liquor potassæ, the fungus will be found to consist of mycelium and spores, the latter, however, being usually very scanty.

The mycelium consists of long, slender, delicate, sharply-contoured pale-greyish ribbon-like formations, or threads, containing here and there spores and granules. It is jointed at

irregular intervals, is remarkable for its length, and usually forms an irregular network. The spores are small round or roundish highly refractive bodies of a greyish color. They are met with singly or in the form of chains, of two, three, or more, and may be isolated or joined to the mycelium.

Upon these microscopic features depends the proof of the disease, though it may generally be easily distinguished by its centrifugal growth, and circular form from the two diseases it is most likely to be confounded with, namely squamous eczema and seborrhœa. This last often takes a circular form on the back and chest, and is associated with some degree of desquamation, but the presence of enlarged sebaceous follicles, and the greasy condition of skin should suggest the correct diagnosis which the absence of the Tricophyton would confirm.

The treatment is simple and easy: wash the part well with soap and water, and then rub on either an ointment of mercury, preferentially the 5% oleate of mercury ointment, or wash freely with a lotion of equal parts of sulphurous acid and glycerine.

Ringworm of the scalp is due to the same parasite as that of the body, but unlike the fungus of Tinea Circinata Corporis, the spores predominate while the mycelium is diminished. It invades the hairs, hair follicles, and epidermis, especially the hairs. The spores are found round the outside of the bulbs and roots, as well as inside the hairs which they completely disintegrate. The hairs become distended with spores, and rupture here and there along the shaft, giving the exterior a rough uneven surface. They become brittle and break, and the free end of the shaft presents a jagged bristly extremity, consisting of broken filaments, between which spores may be found. The epidermis is affected in a lesser degree, and becomes grayish and branny, resembling somewhat eczema squamosum or seborrhœa of the scalp. This latter, however, does not affect the hairs, and should not be mistaken for ringworm.

An error in diagnosis ought not indeed to be possible, for a microscopic examination of the hair must settle the question. In eczema the hairs are not broken or jagged, and do not, as in ringworm, come out easily; and alopecia areata, in which patches of baldness occur, is distinguished by the smooth and shiny surface, while there are never present the broken brush-like hair, which characterise ringworm.

The real difficulty of ringworm is its treatment, and that

because the fungus penetrates so deeply beneath the surface that we cannot reach it, or only more by accident than design.

The disease may last for years, and then seem to get well of its own accord; for this reason there are any number of specifics, the last remedy which happened to be used at the time of recovery winning a reputation only to lose it when tried on a recent case. There ought to be no insurmountable barrier against getting at the fungus, and though in the face of what I have just said, it would be boldness indeed to claim to have discovered a cure, the following method has proved with me more successful than any other. The hair is cut close to the scalp, and then I wash the head well with ether to dissolve the fatty matter of the scalp, which opposes absorption of the drug and its soaking into the hair follicles; and then with a sponge I rub well into the affected part equal parts of ether, glycerine and sulphurous acid. This is done every morning, and every evening a pomade of glycerine of carbolic acid is used for the whole head.

This is the method I employ in cases as they usually present themselves to us, more or less chronic.

If one can get a case in the earliest stage when only the epidermis and perhaps a few hairs are affected, the hairs should be pulled out, and Iodine, which is an excellent parasiticide, painted on the part. Now and then we can by such means prevent the disease extending. It would be impossible to enumerate the varieties of treatment, but I would draw your attention to a method which Dr. Harrison, of Bristol, has found very successful.

Into a solution of half an ounce each of liquor potassæ and spirits of wine, and half a drachm of iodide of potassium, he dips a pledget of lint, which he applies to the scalp for three or four minutes at a time. When this has been done two or three times at intervals of two or three days, he uses a solution of four grains of mercuric chloride in equal parts of spirits of wine and water to make an ounce. This is applied also every other day for a week. He then discontinues all treatment, except inunction with cocoanut oil for a few days, and recommences his former treatment, only using the potassium solution once. He claims for this treatment that it softens the hairs, and permits the formation of biniodide of mercury, where it is most wanted, at the hair bulb itself.

CLINICAL LECTURES

ON

The Varieties and the Treatment

OF

BRONCHITIS.

BY

DR. FERRAND,

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PREFACE

I PROPOSE in these lectures to study the varieties of bronchitis. I will endeavor to present not a deep study of each type of bronchitis, but rather to make a methodical classification of the numerous varieties of the diseases of the bronchi.

The recent discoveries of micro-organisms, while enriching the science of medicine, have not as yet furnished well determined practical applications, and have led to no effective therapeutic indication in this class of diseases.

But here, even as in case of the varieties of phthisis, a rigid scrutiny of different morbid species is fruitful in practical applications, whether it be in the line of diagnosis, or of therapeutic indication appropriate to each species.

My lectures have always been conceived with this end in view, and thus it is that these lectures on bronchitis are the true sequence of those on phthisis. They have been reported by one of my old pupils, to-day our distinguished confrère, Dr. Tisné, and they have been revised by comparison with my notes.

September 27, 1887.

CLINICAL LECTURES ON BRONCHITIS.

LECTURE I.

ANATOMY AND PHYSIOLOGY OF THE TRACHEA AND THE BRONCHI.

BEFORE studying in detail the varieties of bronchitis, it seems essential to recall, briefly, the anatomy and the physiology of the trachea and the bronchi, that is to say, of the tubes through which the air we breathe reaches the lungs.

The continuation of the larynx, the trachea, descends in the anterior portion of the neck and enters the thorax at its superior opening. At about three centimetres from its inception, the trachea bifurcates into a right and a left bronchus, and these extend a trifle obliquely from within without and from above below, the right bronchus being of slightly greater calibre, a little shorter, and lying not quite so obliquely as the left. Each bronchus divides in turn, forming a sort of tree, the branches of which become more and more slender and are scattered throughout the lungs, ending in the pulmonary alveoli.

The shape of the trachea is that of a cylinder with flattened posterior surface, and the larger bronchi gradually lose this flattening until they become perfect cylinders.

The bronchi are in relation with important organs: to the right, the azygos vein; to the left, the point where the aorta crosses the œsophagus; both the bronchi are surrounded by lymphatic vessels and ganglions, and by nerve filaments belonging to the cardio-pulmonary plexus. At the level of the hilus of the lungs the bronchi are in relation with the pulmonary arteries and veins.

We must dwell somewhat on the structure of the bronchi

in order to understand how an irritant may cause the varieties of lesions which are met with. At the outset, we find incomplete cartilaginous rings, the object of which seems to be to hold the bronchi open. In fact these rings become the more incomplete the further the bronchus from the trachea. At first they are like a circle which has lost a part of its circumference, and this loss becomes greater and greater until, when we reach the capillary bronchi, the rings have entirely disappeared. The cartilaginous rings are united together by a fibrous membrane which extends from the larynx. This membrane contains smooth muscular fibres, which are transverse and re-enforce in particular the places where the cartilaginous rings are incomplete. Many of these fibres are inserted at the extremities of the cartilages, forming with them imperfect sphincters, as it were, the relaxation and contraction of which modify the calibre of the bronchi, to the extent of producing a species of peristaltic motion in them.

Inside the bronchi are lined by a mucous membrane which it is important for us to study with care.

The bronchial mucous membrane, like all similar membrane, is finely reticulated, containing within its meshes numerous glands. It is surrounded by elastic fibres, here and there, in quite thick bundles, and it is covered by a layer of epithelium. The cells of this epithelium are cylindrical and ciliated. The physiological action of these ciliæ is most important, seeing that the migration of the products of the bronchial secretion is thus facilitated.

Where two bronchi open into one another, a species of spur is found, which is formed by a projection of the mucous membrane. This spur constitutes an incomplete valve, and it has a tendency to check the passage of the products of secretion.

As we descend from the larger towards the capillary bronchi, the structure alters. The different elements grow less pronounced; the cartilages disappear entirely; the muscular fibres gradually cease to exist; the glands are finally no longer to be found, and the mucous membrane forms simply a thin veil, separating scarcely the aerial channels from the vascular.

The vascular supply of the bronchi differs from that of the lungs. The bronchial arteries are branches of the aorta and of the intercostal artery. These vessels penetrate the muscu-

lar as also the mucous tunic of the bronchi. The veins empty into the pulmonary veins, and here the two vascular systems, pulmonary and peripheral, become one. Finally, numerous lymph vessels follow the bronchi and empty into the ganglions, which are called bronchial because they surround the bronchi.

The bronchial nerves are derived from the anterior and the posterior pulmonary plexuses—that is to say, some are derived from the great sympathetic and others from the pneumo-gastric. These nerves extend both into the muscular and the mucous layers of the bronchi.

Finally, this system of canals has a main function, even as has every living organ; it receives the air from without in order that it may come in contact with the blood. Each of these elements, air and blood, is contained in separate channels; by osmosis the air penetrates the thin walls of the pulmonary alveoli and thus reaches the vascular network of the lungs. The bronchi, therefore, are the carriers of air into the economy—that is to say, they are the scene of hematosis. These physical phenomena, this bringing of inspired air and expelling of expired air, are regulated by a double nervous supply, the one seeming to be the direct cause of the movement, while the other moderates it. Such, indeed, appears to be the *rôle* played by the great sympathetic and the pneumogastric in the mechanism of respiration.

In order to study the varieties of bronchitis, it is necessary to classify them, to unite them into natural groups, even as it was essential when we were considering pulmonary phthisis. This classification can depend on only the three distinct bases on which rests all pathology: the anatomical characteristics, the nosological characteristics, the physiological or pathogenetic characteristics.

An anatomical basis will not suffice, because it is too comprehensive or too elementary for a system like the bronchial. It is too comprehensive, because if we study the effects of irritation on the apparatus in its entirety all grounds for division disappear, and if we endeavor to study it from the standpoint of the anatomical elements which enter into the composition of this apparatus, the multiplicity of these elements would lead to multiplication of varieties of bronchitis, as confusing as it would be artificial.

A nosological basis will not answer here, although it did in case of the forms of pulmonary phthisis. In case of phthisis we are dealing with a definite morbid element, better understood to-day than ever, on which depends every tubercular process. It is in the variety of the causes which lead to this element that we must seek the basis of a classification. In case of bronchitis matters are different. The element is the cause, so to speak, I mean the immediate cause which yesterday was called irritation, and which possibly to-morrow may have the same name. This irritation gives rise to one or another symptom according as it determines in the bronchial apparatus one or another nutritive modification, and according as it ends in altering the vascularization, the secretion, the intimate nutrition, and finally the innervation of the bronchi.

The following, indeed, is the only practical classification of the varieties of bronchitis: Congestive bronchitis; Catarrhal bronchitis; Inflammatory bronchitis; Spasmodic bronchitis.

As for the diathetic variety, we will not neglect it, but I will point out under each variety in what manner it is connected with this or that constitutional disease, and how this or that diathesis is especially prone to be associated with one or another variety of bronchial irritation.

LECTURE II.

CONGESTIVE BRONCHITIS.

PROBABLY the most simple form of irritation of the bronchi is the congestive.

A lesion so elementary as congestion, so variable, so readily confused with other lesions, cannot always be readily separated from these others. Congestion consists in afflux of blood. In the bronchi it is afflux of blood into the mucous membrane, which we have seen is both so thin and so vascular. Such, indeed, is the characteristic of the mucous membrane throughout all the air passages. You doubtless will recall how slender and vascular the pituitary membrane of the nasal fossæ is and how readily it becomes congested.

You will the more readily understand the effects of congestion of the bronchi when, having told you in what it consists, I tell you in what it does not; that is to say, how it is differentiated from both the catarrhal and the inflammatory lesion.

Although we will plainly perceive the truth of the following in the course of our study, we desire to emphasize it now: congestion is sharply differentiated both from catarrh and inflammation. Vascularity plays the only *rôle*, and the afflux of blood is at the basis of it. While catarrh depends in the main on a disturbance in the secretory function and ends in a modification, above all quantitative, of the products of secretion which are constantly on the increase, congestion does not increase secretion, but on the contrary diminishes it, and at times even suppresses it, especially at the outset. While inflammation, by altering the nutrition of the elements involved, infiltrates them with a new exudation and leads to granular and to purulent degeneration, congestion respects these elements, simply irrigating them to excess, and allows them, as soon as the afflux has ceased, to regain in thorough integrity the functions proper to them.

The difficulty met with post-mortem in delimiting these

various morbid processes, even by the aid of the microscope, has resulted in their being deemed nearly similar even if they have not been deemed one and the same. I hope to prove to you that clinically they ought and can be differentiated, and that such a differentiation is of the greatest possible importance both from the standpoint of prognosis and of therapeusis, that is to say, from the standpoint of what constitutes the chief difficulty and merit of a sound diagnosis.

A further distinction, deemed of too little note to-day and yet most essential clinically, is the differentiation of active from passive congestion. Under the plea that these two forms of congestion are often so intimately associated as to be practically inseparable, the tendency is to no longer differentiate them, a tendency which for more than one reason is to be regretted. A common factor in both of these processes is exaggerated afflux of blood; but in case of active congestion the afflux is the result of the passage of an excessive quantity of blood in a given time through the vascular system which is the seat of congestion; and in passive congestion there is accumulation and stasis of a greater quantity of blood in this system. In a word, active congestion consists in a hyperemia through excessive activity of the circulation, and passive congestion is hyperemia with stasis. The latter is above all the result of mechanical obstacles to the circulation; the immediate cause often lies beyond the locality where it is produced, in the heart for instance; the former is usually connected with some form of irritation acting on the organ itself; it appears to result from an appeal for blood from the irritated organ itself, as it were. In reality it follows on a localized vaso-motor dilatation of the vascular system of the organ.

Do not wonder that I dwell on these distinctions, which are far too frequently misinterpreted or not recognized. You will shortly better understand how important it is to recall these facts to you.

We will in particular speak of active congestion, in the first place because less is known about it, and secondly because it is important to the physician under the two forms, acute or chronic, under which it is manifested. Finally, bronchial congestion must be subdivided according as its seat is in the larger or the smaller tubes. In the first case, the affection is ordinarily not serious, although not always mild in its

course; in the second, when intense and persistent, especially when general throughout all the bronchial ramifications, the entrance of air into the capillary bronchi may be entirely prevented, the respiratory field in proportion diminished, and the patient be subjected shortly to the risk of true asphyxia.

Symptoms.—The symptoms suggestive of congestive bronchitis consist in subjective and in functional troubles.

The first sign is a peculiar sensation of fullness accompanied by heat and even burning in the chest. These sensations are at times limited to the retro-sternal region, and often this same region is the seat of true pain, permanent, a pain which is awakened in particular by forced inspiratory movements, efforts at cough or at sneezing. This pain may extend to the base of the thorax, near the false ribs and around the waist.

These sensations are accompanied by functional disturbances, and, at the outset, by more or less aggravated dyspnea. The reason for this dyspnea is apparent. The thorax is a cavity the capacity of which varies with each respiratory cycle; when it dilates, the air and the blood tend to rush in in order to fill the virtual vacuum which follows on the enlargement of the cavity; the entire intra-thoracic space which is set free must be filled either by air or by blood. Now when the blood flows into or remains in this apparatus, it occupies a portion of the space to which the air has a right. Thus it is that congestion diminishes the quantity of inspired air, curtails the field of hematosis and causes dyspnea.

This dyspnea is accompanied by cough, the characteristic of which is unique enough to deserve special mention. The cough is dry, repeated in series, painful, tiring, increasing the engorgement of the thorax instead of relieving it. It causes no expectoration or else results in the expulsion of a slight amount of glutinous mucus, occasionally a trifle discolored by blood. At times, a spasmodic element being superadded to the congestion, the cough becomes more distressing, even to the extent of producing vomiting, which is, of course, reflex in nature. This occurrence, however, is rare and is not met with in simple bronchitis.

On auscultating the thorax, the respiratory murmur is rough and dry all over; there are few râles or only a few sonorous, varying in tone according to the depth of the congestion, loud in the large bronchi and sibilant in the small. In

case the congestion is intense, the respiration ceases to be roughened, but becomes obscure and the murmur may even disappear entirely; it is then that the swollen bronchi diminish in calibre, so much even as to prevent the ingress of the column of air.

Aside from the signs yielded by the bronchial apparatus, properly speaking, there are others from the side of the neighboring organs. The mucous membrane of the upper respiratory tract, which is in such intimate functional relationship with that of the bronchi, sympathizes always with affections of the latter. This is the reason why in case of congestive bronchitis we observe congestion of the pharynx as also of the larynx. Therefore the presence of a certain degree of erythematous angina, readily discernible, and of congestive laryngitis which is marked by simple bronchitis and by change in the voice.

As for the lungs the same negative signs are present; there is neither murmur nor dullness, that is to say, but a simple diffuse roughening of the respiratory murmur; at times the respiration is choked when the congestion is intense. When râles are present, they consist in short whistlings produced in the bronchi of mean or of fine calibre.

The general signs of congestive bronchitis, finally, are very variable, and seem to depend, at the outset, without doubt, on the extent and on the acuteness of the congestion; but also, and especially, on the surroundings amid which it developes, and, further still, on the general health of the patient. This is why usually pure bronchial congestion is almost entirely afebrile, while the form which accompanies colic is febrile of high enough degree, and while, further, the form which accompanies emphysema in certain patients, or that which occurs in children, is often attended by great elevation of temperature.

LECTURE III.

VARIETIES OF CONGESTIVE BRONCHITIS.

THIS lecture is the most important of all in our study of the varieties of bronchitis, and from it will result indications of the most practical nature.

Simple Congestive Bronchitis.—The most simple form of the affection is that which we witness so often, so frequently, indeed, that it scarcely arrests our attention. The fact is that its mild course and its frequency will account for the manner after which it is neglected.

In determining the most efficient cause we must consider two elements: the one due to atmospheric changes, to variations in temperature, natural or artificial, in a word, to all causes which lead to chill; the other depending on a peculiar impressionability of the integument, which is further often in accord with a constitutional predisposition of the patients.

This manner of action is explained by the direct and topical irritation of the cold air on the mucous membrane of the bronchi, and also by the intimate physiological relations which exist between the skin and the bronchial mucous membrane. These two respiratory surfaces are both directly subject to atmospheric contact, and send the one to the other the impression which they receive, and what is more astonishing still, is the fact that the impressions on the skin may react on the bronchial mucous membrane, whatever the part of the integument which has been exposed to the external influence.

This congestive bronchitis is ordinarily a simple cold, which the physician should recognize as being the first type of a pathological series, but which rarely requires his interference, and disappears in a few days, often in a few hours. When matters happen differently then it is because a foreign element has become superadded, and then we have to deal with one of the varieties which I am going to describe.

Congestive Bronchitis by extension from the mucous membrane of the upper air passages.—We have here to deal with the whole category of the congestive irritations of the naso-pharyngo-laryngeal mucous membrane, which may by extension complicate congestive bronchitis. The physiological relation which subsists between all these mucous surfaces explains sufficiently the method of extension, and how coryza, pharyngitis, and congestive laryngitis may easily become a bronchitis of the same morbid nature. But this form need not further claim our attention any more than the preceding. The reverse holds true for the following form.

Congestive Bronchitis of pulmonary origin.—Owing to the continuity of surface existing between the ultimate ramifications of the bronchi and the pulmonary alveoli, it is apparent how irritative affections of the lungs are accompanied by a certain degree of irritation of the bronchial mucous membrane; there are certain varieties, however, which are specially prone to this complication. These we proceed to study.

A. At the outset, *whooping cough* and *asthma.*

In case of whooping cough congestion accompanies the first stage of the affection; it leads to the dry cough, not specially characteristic; there are no râles to be heard, but only roughening of the respiratory murmur.

It is established that the general depression which exists during whooping cough, and in case of confirmed asthmatics, exposes them especially to morbid affections, and in particular is the integrity of the respiratory apparatus and of the bronchial mucous membrane at stake. Each asthmatic crisis as well as that of whooping cough causes sharp oscillations in the tension of the bronchi, which necessarily react on the circulation of these tubes. Congestion thus accompanies the crisis, and it tends to persist in the capillary system of the pulmonary vessels, worn out, if not paralyzed by the repeated or sustained tension to which they have been subjected. Thus it is that often the bronchial congestion, at the outset transitory and following only on the spasmodic attacks, becomes more persistent and eventually permanent.

We must not, however, forget that the congestion of the bronchi, nere secondary, may play the *rôle* of a provoker of the asthmatic attack when it occurs accidentally, as it were, or independently of the actual course of the affection. When-

ever secondary, however, and this is most frequently the case; it is passive in a certain sense, although it is dependent on a state of excessive excitability of the pulmonary innervation. I will return further on to this point.

The effect of irritant causes, too dry or too cold an atmosphere, dust of various kinds, etc., are without question frequent sources of bronchial congestion; but the variety of greatest interest is that which follows upon the crisis. The difficulty with which the air is renewed in the chest during the crisis results in an afflux and a stasis of blood. It is this afflux of blood to the mucous membrane which, after having played a part in the dyspnea, starts the catarrhal period by apportioning to the glandular and epithelial elements the materials indispensable to the secretion which is going to exist after the access and give the patient rest.

B. In *pulmonary emphysema* there exists no tendency to congestion of the parenchyma of the lung. Indeed the emphysematous lung becomes slowly changed, so that the separate alveoli are transformed into simple cavities, of greater or less size, in which the radicles of the pulmonary vessels lose in great part their expansile surface. But, if the vesicular element is atrophied, and with it the pulmonary circulatory system, the same does not hold true of the bronchial element; the bronchi dilate, and their vessels, in accord with this same hypertrophic movement, develope to excess. This is the reason why emphysematous patients are so often exposed to bronchial congestion.

We have over and again pointed out to you in the wards emphysematous individuals in whom, under the influence of some cause, but slightly irritant, the bronchi become engorged, so that the dyspnea is ardent and the cough short and choked, frequent and without much expectoration, perhaps a little stringy mucus more or less mucilaginous.

At the same time, scattered over the chest, sibilant râles are heard, short in duration and occurring only at the end of inspiration, and a few sonorous râles accompanying expiration; while coincidently with these râles, and particularly at the apices, the vesicular murmur is heard with the moist ring which is proper to emphysema. These phenomena are not only associated with dyspnea, at times excessive and even reaching orthopnea, but the respiratory anguish is often in-

creased by the pains which the patient has in the thoracic walls, notably around the waist and at the level of the attachments of the diaphragm.

The mucous membrane of the upper respiratory tract takes part in these disturbances; a pharyngitis results which is marked by gurgling and by general redness of the mucous membrane and of its papillæ, and later by permanent granulations. Change in the voice is a sign that the larynx is in turn affected.

This pathological scene may be played without the occurrence of much fever. Often, however, the temperature rises, the appearance is congested, the perspiration abundant, the pulse strong and frequent.

At other times, to these phenomena of fever with congestion, are superadded those of asphyxia, cyanosis, lividity.

The phenomena do not often attain such intensity; they often decrease in degree and then return with ease. Sooner or later their repetition leads to permanency, and the paroxysms are no longer characterized by an acute congestive crisis, but there exists a stasis which is intensified by the progressive dilatation of the heart, which promotes the asphyctic phenomena to which I have referred.

Finally, there is a certain type of congestive bronchitis on which I cannot insist too strongly, because it is generally not detected, oftener confounded with emphysema, or with pulmonary congestion, and because further it constitutes a morbid entity as painful as it is refractory to therapeusis. (*Vide* the thesis of my pupil Dr. Chuquet.)

I cannot better describe this type than by relating the pathological history of the late janitor of this school; he was a robust man, about thirty-five to forty years of age, of great breadth of chest, high color and very muscular. He had suffered from certain vague manifestations of rheumatism, with the nature of which I am not familiar any more than I can tell you in what degree alcoholism played a *rôle* in his case; sufficient the statement that one or the other of these conditions is generally of much account in the etiology of the phenomena which are claiming our attention.

This man, whose occupation did not expose him to changes in temperature, was nevertheless almost always with a cold, and was markedly emphysematous. But this would not have

troubled him particularly if he had not been seized every now and then by attacks similar to those of which we are treating. Three to four times each year, and the attacks became more and more frequent, he was subject to a violent spasmodic cough accompanied by a species of suffocation, intense congestion of the face, exorbitism and lachrymation—in a word, all the phenomena of an attack of spasmodic cough and anguish the most unendurable. These attacks were associated with only slight expectoration, a little stringy aerated mucus, at times striated with blood or even with pure or dissolved blood in greater or less amount.

On examination, the pharynx was intensely congested and studded with the remnants of old granulations of the mucous membrane. The nasal passages were dilated and the voice deeply altered. The respiratory murmur was veiled by numerous sonorous, vibrating râles varying in quality; at the base of the lungs a few sparse mucous râles could be heard. The patient had sufficient fever to oblige him to keep his bed, aside from the fact that the dyspnea and difficulty in breathing which marked the attacks practically incapacitated him. Concomitantly all alimentation was suspended.

After having tested in vain the routine therapeutic measures, blisters, etc., I concluded to test emetics. Owing to the decongestinating action of ipecac, I ordered it, but without much effect. Convinced, however, that such was the indicated therapeusis, I resorted to a more powerful drug, and ordered tartar emetic. The first day the patient took .05 grammes, the second day the same dose, and from the third day on .10 grammes. The nauseated condition into which the patient was thus thrown was so distressing that I did not continue it more than a few days. The patient was so much relieved that he took the drug for about a week, at the end of which time the attacks had ceased.

We might think that the attacks would in this time have ceased of themselves, although the drug may have hastened matters somewhat. My answer is that ordinarily the attacks when left to themselves lasted from six to eight or more weeks. Finally, and this is curious and important, the patient, the attack once at an end, breathed more freely than it had been possible for a long time, being able to walk and to work with far greater ease. Let me add that the recurrences became

less frequent when the man obtained a better position and then he passed from under my observation.

To cite another case:—a grocer, after repeated attacks of bronchitis, ended by having the same pharyngo-laryngitis, losing his voice almost entirely and suffering from spasmodic attacks like those just described. The man was strongly built, rather stout, and yet was at times confined to his room for weeks, during which he was subject to very severe paroxysms, accompanied by phenomena of suffocation and of asphyxia.

The same treatment was prescribed by my advice by his family physician, and to-day the man is well.

Still another instance is that of a civil engineer, who, by his occupation, is subject to great changes of temperature, often travelling at night, and is considered as having a serious affection of the lungs. When I saw him he presented the same symptomatic look: granular, congestive pharyngitis, chronic laryngitis, frequent attacks of congestive bronchitis, and the patient actually thought of renouncing his occupation. After the treatment I have outlined, the attacks disappeared, and this man pursues his profession in a state of sufficient health.

I might further refer to a missionary, arthritic and very fat, in whom fatigue and defective hygiene have resulted in fibrous rheumatism, emphysema, cardiac dilatation to a certain extent, and attacks of congestive bronchitis with the attendant phenomena. The same treatment resulted favorably, although the condition of the heart did not allow as complete a cure as in the previous cases.

I have deemed it essential to insist on these points because we are speaking of a pathological variety not referred to in your books, and yet which you will meet with in your professional career, and also because the cure is difficult and the only efficient therapeusis which I have found is that of which I have spoken.

C. Pneumonia.—There is always present in the neighborhood of a pneumonic centre a zone of pulmonary congestion around which the bronchial mucous membrane is itself more or less profoundly affected. Now this bronchial congestion is veiled by the pneumonia and plays a secondary *rôle*. But before the pneumonia is pronounced it is not rare to find re-

peated bronchial irritation for a number of days. Still more frequently do we note the tenacity, sometimes extreme, of the bronchial irritation which at times persists after convalescence. This irritation, it must be noted, depends rather on a diathesic condition than on the effect of the bronchial congestion itself.

Finally, it may happen during the course of a frank pneumonia, that the patient is seized by extreme dyspnea, without any increase in the inflammatory area, a dyspnea which is then the result of a bronchial congestion quite intense, and generalized enough to compromise life. We are dealing with a condition independent of the pneumonia, with a newly superadded agent, which calls for special treatment; in these cases venesection triumphs over the asphyxia by decongestioning the bronchial mucous membrane. This fact is well proved by a case that I noted in the *Bulletin de Thérapeutique.*

D. Tuberculosis.—You are aware that pulmonary congestion is a frequent accompaniment of tuberculosis, that it often precedes it, that it always accompanies it to a greater or less degree, and that it increases at the periods of change of the tubercle, when it degenerates, for instance.

Congestive bronchitis is possibly less frequently met with, but this variety is often found in the tubercular. It is often the premonitory sign of an imminent or beginning tuberculosis. It is it which causes in many young people that distressing, tiresome, even painful, dry cough, without expectoration, which should always direct the attention of the physician both to the existence of congestion, and to the cause at the bottom of it. This cough and the congestion at the base of it are intensified, besides, at each addition to the tubercular process, and are accompanied by more rapid respiration or even by true dyspnea.

When the tubercular foci break down, congestion is again an accompaniment, but it extends less to the surface of the bronchi. The focus which softens, even as the exudation which suppurates, provokes less irritation in its neighborhood than it does during the formation stage. The bronchitis then tends to pass from the congestive variety to the catarrhal.

Finally, the bronchial congestion remains permanent around the tubercular foci, becoming confounded with the pulmonary congestion, and the two processes together are

responsible for the various abnormal sounds which may be heard in the neighborhood. But, even as happens in case of pulmonary congestion, there is formed a peri-tubercular zone in which are multiplied the elements capable of contributing to the propagation of the lesion.

Enough has been said to prove how essential it is to fight this process, which constitutes of itself a new source of suffering, and which assists so powerfully in the genesis and in the extension of the disease.

I was recently able to show you two patients presenting these symptoms, and in whom you had an opportunity to witness the amelioration under the influence of a medication emollient and derivative. I might tell you, in addition of a woman, the inmate of a religious community, who recently came to Paris. She has been called tubercular and so treated, having been ailing for two months, by every routine means which suggest themselves. This patient was subject to frequent and to irregular febrile attacks, coughed for hours, the cough being of the dry, irritant nature, of which I have just spoken, suffered from insomnia and almost total loss of appetite. At one of the apices there were marked râles, and in the rest of the lung a few vibrating râles unequally disseminated in the bronchi, and grouped especially near the apex. Directing my treatment towards the bronchial congestion which I recognized, I stopped all balsamic or exciting medication; I ordered a little antipyrin, emollient fumigations, poultices over the thorax, a little opium, and in two days all the symptoms had disappeared (except, of course, the râles at the apex); the patient was able to resume her journey, scarcely believing that she was not cured, but in reality only relieved of an accidental complication. True enough this complication was a serious matter in itself, and might speedily render fatal a disease which, thus ameliorated, may remain quiescent for an indefinite time.

E. Other Organic Affections.—Around tumors which may develope in the lungs, bronchial congestion often occurs connected with the growth of these tumors, softening of cancer or of gummata, rupture of cysts, etc.; or rather, these foreign bodies act as a predisposing cause in virtue of which the most simple occurrences become causes of bronchial and of pulmonary irritation.

F. Pleurisy.—Pleurisy constitutes a better determined cause and a more interesting one of congestive bronchitis. This affection ordinarily is preceded by a dry, distressing cough, analogous to that of congestive bronchitis, being accompanied by a sensation of oppression in the chest, especially retro-sternal, which we are justified in considering due to an active congestion of the bronchial mucous membrane.

Later, when the exudation has become absorbed and has been replaced by a more or less solid layer of false membrane, the same cough may occur and is due to the congestive irritation of the bronchi. But then the congestion is apparently associated with a peculiar blood stasis, the fibrous shell which surrounds the lobes corresponding to the lungs, opposing the free entrance of air as well as the elastic play of the lung itself.

Congestive Bronchitis of Indirect Origin.—Under this heading we will make the following subdivisions:

1. *Affections of the Heart.*—Aside from affections of the pulmonary system, diseases of the heart lead most frequently to bronchial congestion. These patients may suffer from active congestion of the mucous membrane. Furthermore, the obstacles in the way of the circulation through the vascular system of the bronchi cause, at least in the beginning, an excitability of the mucous membrane which easily results in colds and in bronchial irritation. This is the reason why such patients derive much benefit by a sojourn during the bad months in a southern clime, amid equable temperature.

2. *Affections of the Stomach.*—In many neuroses of the stomach and even in the beginning of a number of organic affections of this viscus, the patients complain of a dry and distressing cough accompanied by a sensation of retro-sternal pressure. It is evident that, especially at the outset, this cough is usually nervous, but not infrequently it becomes congestive. Besides, were it not such at the beginning, it would soon become so through the irritation which constant repetition causes.

3. Affections of the *liver* and even those of the *kidney*, may play a similar *rôle*. For the latter organ in particular, bronchial irritation may not only be sympathetic or reflex, but it may also follow on the congestion due to the elimination by the bronchi of a certain proportion of the ammoniacal ex-

cretions which the kidneys do not dispose of, or insufficiently so in case of inflammatory affections of the organs.

4. *Acute diseases of the skin* are often complicated by congestive bronchitis. Erysipelas, for instance, may lead to this complication. It is more common, however, in most of the eruptive fevers. Among them, measles is most likely to provoke bronchitis; but this is more frequently catarrhal than a congestive bronchitis. Often, true enough, the latter becomes the former.

5. In case of *typhoid fever* there is generally at the beginning a certain degree of irritative and congestive bronchitis.

6. *Menstrual disturbances, puberty, pregnancy,* and the *menopause* are often accompanied by bronchitis. Whether the congestion be supplementary or complementary, the mechanism is readily understood, and I will not dwell upon it. It is only later on that the congestions arise which are the result of the difficulty with which the blood circulates, or to morbid changes in the blood.

Congestive Bronchitis due to the Elimination of Medicinal Agents.—An interesting form of irritative congestion of the bronchial mucous membrane is that which is due to the elimination of certain medicinal substances, and this explains the calmative action of such agents as the balsams, sulphur, creasote. These agents, introduced into the blood by absorption from the intestinal canal, come with the blood into contact with the bronchial mucous membrane, and are thence eliminated by means of a species of transudation, leaving behind a greater or less irritation. This irritation of the respiratory mucous membrane is seen very frequently after the ingestion of the iodide of potass, and, better still, after various balsams. The knowledge of this fact should warn us against the premature use of these agents in case of broncho-pulmonary congestion. Indeed, except in the torpid forms where the favorable effect is due to the excitation of the mucous membrane, their administration often results in a marked aggravation of the irritability of the mucous membrane.

Diathetic Congestive Bronchitis.—Rheumatic and gouty patients, more so than others, may present the common variety of bronchial congestion, but there occurs in them a special variety, interesting on account of its localization. It occupies the borders of the lungs, in particular the region above the

sternum, and is accompanied by fine and sharp râles; this *marginal bronchitis* is only a localization of the congestions to which these patients are so prone.

A more interesting variety still is the dry bronchitis of the gouty. It exists aside from the attacks, the cough being dry and sonorous, without secretion, and being spasmodic. This irritation, awakened by cold and by dry heat, is very painful, and it ends often enough in catarrhal bronchitis.

Further causes of congestive bronchitis are age, the torpid variety being found most frequently in the old, while in the child the affection is more acute, and tends to merge rapidly into broncho-pneumonia. In children, also, dentition is one of the most frequent causes of congestive bronchitis.

In a word, any morbid element exposes the subject to congestive manifestations, and therefore to congestive bronchitis. After the analysis we have made, it is impossible without repetition to insist on further causes.

LECTURE IV.

CONGESTIVE BRONCHITIS—PROGRESS AND DIAGNOSIS.

CONGESTIVE bronchitis in its simple form is nothing more than what is ordinarily called a cold. It may last for days or for many weeks. The general febrile phenomena are often marked at the beginning, and again slight and transient, not sufficient to cause the patient to stop work, ending soon with or without perspiration.

Ordinarily the congestive variety is somewhat modified, and the affection is altered into a more or less abundant secretion, into a catarrhal expectoration, which, as I have stated, sometimes follows on the disappearance of the general symptoms, and is always accompanied by a feeling of deep relief.

Congestive bronchitis often affects the *intermittent* variety, possibly because of the changes in the general, and in particular, in the pulmonary circulation. Thus certain patients feel better towards night, and others become notably worse—a change for the worse which not only concerns the local symptoms, but also the general, such as the elevation of temperature, the pulse rate, etc.

At times the type is *intermittent*, the local and general symptoms recurring at a certain hour. This holds more especially for malarial regions, and during certain epidemics of congestive or catarrhal bronchitis.

Finally, I must refer to the recurrent attacks of congestive bronchitis so frequent in infants and in the old, and in every individual at times of special predisposition, such as the menstrual periods, pregnancy, the puerperal state, or in those the subject of heart disease or emphysema.

Such foremost is the congestive bronchitis on which I have laid special stress, as occurring particularly in emphysematous rheumatics. I then noted how this variety of bronchitis was paroxysmal during the attacks, and recurrent on the slightest provocation.

A grave variety, frequently conjoined with the preceding, is *suffocative* congestive bronchitis, rarely met with in the adult, but often in the infant and the old. This form is almost always consecutive to an organic cardiac, pulmonary, or renal affection. I have seen it occur during the course of scarlet fever and of measles.

In case of measles it is rarely suffocative of a sudden, and occurs usually by propagation of the congestion from the larger to the smaller bronchi. In case of scarlatina it may declare itself more abruptly.

I recall the case of a little boy suffering from an ill-developed scarlet fever. Instead of convalescing normally, he remained in a precarious state, although without special symptom, so much so that a pædiatricist, whom I called in consultation, gave a relatively good prognosis. Two days thereafter a congestive suffocative bronchitis declared itself, from which the patient died in a few hours. I should add that there existed only an insignificant quantity of albumin in the urine.

The urgency of the symptoms may awaken the belief that we are in the presence of an attack of asthma or of a suffocative catarrh. But the sibilant râles and the absence of expectoration proves that the congestion of the mucous membrane is intense enough to render it turgid to the extent of almost obliterating the lumen of the bronchial tubes, and of preventing the access of air into the lungs. Whence follows a suspension of hematosis, and a condition of asphyxia which may prove fatal.

Congestive bronchitis does not always occur under an acute form. We have seen that while certain varieties of the affection are very transitory, others are very prone to recurrence, and in consequence of repetition the affection tends to assume a chronic type.

Especially in connection with the diatheses does bronchitis assume the latter type; witness the occurrence in rheumatic and in gouty individuals. Dry bronchitis, marginal bronchitis, recurrent bronchitis—such are the varieties most frequently met with in these individuals, and the marginal form usually becomes chronic, particularly in the rheumatic. In the gouty we most frequently find dry bronchitis, and this is rarely chronic in type; at least, when congestive bronchitis

becomes chronic in the gouty it is usually the result of repeated, successive, prolonged acute attacks.

Finally, whether the individual be rheumatic or gouty, it is in connection with these affections that we find the variety of recurrent congestive bronchitis, to which I will return in particular under the head of treatment.

In case of scrofula, bronchitis is certainly not an uncommon accompaniment; but it is not the congestive type which it ordinarily affects; we will see the truth of this when we speak of catarrhal bronchitis. Still, exceptionally, in the scrofulous we meet with congestive bronchitis as the result of some accidental cause such as is likely in any event to give rise to it.

Diagnosis.—Congestive bronchitis is characterized, we have said, by: 1. A subjective sensation of retro-sternal pressure. 2. An objective dyspnea out of proportion to the physical signs of the affection. 3. The absence of abnormal sounds, which are usually replaced by a roughening of the respiration, or purely, even in the aggravated forms, by whistlings, and even by absence of murmur, or other sign indicating a modification in density of the pulmonary parenchyma.

It is above all important to differentiate carefully congestive bronchitis from catarrhal bronchitis. If you recall the difference we have stated between congestion and catarrh, it is an easy matter to understand the signs by which these two forms of bronchitis differ.

Catarrhal bronchitis is accompanied by a sensation of retro-sternal difficulty much less accentuated, often none at all or insignificant, by less dyspnea, dependent, however, on the greater or less extent and depth of the lesion. Finally, and above all, the secretion is more or less abundant, and consequently there are present mucous râles of varying calibre and an abundant fluid expectoration; while congestive bronchitis produces only an insignificant mucous secretion and an expectoration, slight or viscous. The diagnosis cannot, hence, be difficult, except in the instances where the two processes, catarrhal and congestive, are combined or follow rapidly the one on the other.

Inflammatory bronchitis must also be differentiated from the purely congestive variety. The inflammatory form is accompanied by a more intense febrile movement, more abrupt and more continuous. As for the physical signs, the abnor-

mal noises, the sibilant and sonorous râles, some mingled with moist râles, are met with in the inflammatory form in quick succession, while they are much more rare in the congestive variety. Finally, the expectoration is always profuse enough at the outset; true enough, it is sparse and mucous, and this approaches the congestive type, but it shortly becomes abundant and muco-purulent, a characteristic of the inflammatory variety of bronchitis.

The fact is that the ordinary rule is for bronchitis to be often congestive before becoming purely inflammatory; this is the reason why generally bronchial congestion is considered the first stage of the inflammatory, and therefore as not deserving special description. I hope that after what I have said you will avoid this unfortunate error and will recognize and treat bronchial congestion as a separate entity.

As for spasmodic bronchitis and the true neuroses, they are differentiated from bronchial congestion by the absence of fever, by the progress of the affection—that is to say, by the intermittency of the attacks and the greater or less regularity of the recurrences, and by the abruptness with which they often begin and end.

It is apparent that here as well congestion often complicates the spasm, and that it is present, as we will see, often as effect if not as cause.

In order to weigh the delicate points in the diagnosis it is essential to consider the general state of the patient; fever is absent in the true and habitual neuroses, at least thoughout the greater part of their evolution. The abnormal sounds, so variable in case of the neurotic attacks, are present persistently in the congestive attacks; and when the signs of these two factors coincide it is because the factors themselves co-exist, and this is not infrequently the case.

From a diagnostic standpoint, no less important a question as regards therapeusis is the determination of the cause, more or less direct, of the congestive bronchitis. It is unnecessary for me to repeat what I have said already in regard to the causes and the varieties of this affection. I only desire to resume the problem by stating the points which are furnished by the extent of the congestion, by its seat, by its evolution.

When congestive bronchitis is of much extent, we ought

at once to think of a general cause. Generalization of the affection allows us ordinarily to eliminate tubercular bronchitis, which affects the apices and cardiac bronchitis which involves the bases. True enough congestive bronchitis may become generalized even as may the morbid product with which it is associated, and, in case of cardiac disease an active and general congestive bronchitis may occur. But it is true also that such instances are exceptions to the rule.

The seat of the bronchial congestion is also of significance. I have mentioned the marginal form met with in rheumatics, which is accompanied by fine, bursting râles, and which is found along the interlobar fissures, in the axilla, above the diaphragm, in particular in the emphysematous.

I need not dwell again on the significance of apex bronchitis.

The progress of the bronchial signs is also of great importance. Bronchitis beginning at the base, and in which the râles as they become more abundant replace the roughening of the vesicular murmur, is a hypostatic form, which may be due to an obstacle in the circulation (oftenest cardiac disease) or to alteration of the blood (typhoid, nephritis, etc.).

In conclusion, the study of the extent, seat and progress of the disease, suffices to establish, with the diagnosis, a strong presumption in regard to the causal factor of the congestion; this presumption becomes a certainty when it is confirmed by other practical considerations drawn from the patient.

LECTURE V.

CONGESTIVE BRONCHITIS—TREATMENT.

THE treatment of congestive bronchitis depends, as does all therapeusis, on a triple indication—that is to say, on the seat, the form and the cause of the affection. In other words the indications are anatomical, physiological and nosological.

The anatomical indication consists here in bringing to bear topical medication directly on the irritated surfaces and on those which are in near contiguity, or else indirectly on the parts which are physiologically or sympathetically in common relation.

An emollient indication is the first which suggests itself among those which are applicable to the affection, since we are dealing with irritant congestive disease.

To fulfill this emollient indication, the first thing to do is to remove all sources of fresh irritation. It is hence essential that the bronchial mucous membrane should be excited as little as possible; and since we cannot remove it from contact with the air, this air must be in composition, in warmth, in purity, in moisture above all, as little irritating as possible. Equability in the composition of the air plays an important part, and so long as the composition does not change, the irritation the mucous membrane is subjected to is slight. It is evident, therefore, how important it is, in even the less serious forms of congestive bronchitis, to keep the patients at home, to cause them to avoid change in temperature as well as impure inhalation, even though the air be only slightly charged with dust.

Not only must direct irritant agents be kept at a distance, but also (and this is very important, for it is a rule often neglected) the indirect irritants—that is to say, all agents that act by elimination. To explain the matter, you know that many hygienic or medicinal agents, once in the economy, are eliminated by the mucous membrane of the bronchi. In pass-

ing through this membrane they exert a modification in nutrition which usually is followed by irritation. Such, for instance, are many gases, carbonic acid in particular, also sulphohydric acid. In addition we must mention the balsams, sulphur, creasote and all its compounds. None of these agents can be tolerated when the indication is for emollient medication. Still they may prove of value when, the stage of active congestion having passed, there remains only a chronic and passive congestion to combat. At the outset these agents increase the ill, and even aggravate it.

Even in case of diathesic patients, when the bronchitis is coincident with constitutional influence, there exists almost always an occasional cause which at least determines the localization of the irritation on the bronchial mucous membrane, and this cause it is above all essential to remove.

An emollient medication is the most likely to fulfill this indication. Soothing drinks, mucilaginous soups, hot or lukewarm, act on the neighboring mucous membrane; further still, their introduction into the blood facilitates glandular secretion, as well on the skin as on the mucous membrane, and in consequence tends to provoke the catarrhal flux which we have seen modifies so favorably congestive inflammation of the mucous membrane and determines its resolution. Fumigations, which are practised with mild aromatics, are more direct agents than the drinks, and constitute one of the most rational of the topical treatments, and are most effectively useful. The moist, warm vapors from the fumigations act as a soothing bath on the mucous membrane, and favor the separation of the altered mucous membrane, and of the slight viscous secretion which has formed on its surface.

The cutaneous surface of the thorax may also to advantage be used for the application of emollient agents. Thus you have seen me apply with great success large poultices of linseed meal on the chests of patients subject to the irritant cough and the retro-sternal pressure of congestive bronchitis. Large plasters applied over the chest or the back have a similar influence, and the same holds true of collodion. Inunctions of fatty or oily substances fulfill a similar indication, and may be most useful.

All these agents, of which the poultice is the type, act by retaining within the tissues the humidity which, normally, is

constantly evaporated from the skin, and also by maintaining on the surface a species of local bath; perhaps also by increasing the conductivity of the superficial tissues. These applications favor the emission of heat, and this frees the tissues from the excess of heat which is altering or rendering them more vulnerable. Whatever the case, whether the action is chemical and there is diminution of oxidation of the tissues, or whether there is a sympathetic modification between the two surfaces, skin and mucous membrane, it is certain that the above agents facilitate the secretion of the bronchi and decongestionate them.

Revulsive medication must often assist the preceding. It is practised on the skin, preferably that of the thorax, and particularly over the surfaces which correspond with the affected bronchi. We know that the anterior and superior regions of the chest are those where the cutaneous sensibility is perhaps most developed, and, in any event, it is the most apt to awaken the reflexes of the respiratory centres, those in a word which, in asphyxia, maintain the longest the power of provoking reflexively the respiratory movements.

This therapeutic method is peculiarly applicable to the cases where deep congestion does not yield or does not seem to intend to yield to emollients. The agents at our disposal are: tincture of iodine, mustard plasters, frictions and by ammoniacal liniments, camphor, alcohol, spirits of turpentine; finally the blister, which is rarely indicated on account of its limited action. If necessary the punctate cautery may be used, but its value is exceptional.

Intestinal revulsives are less frequently utilized in these forms than in the others; nevertheless the slightly soluble or insoluble antimonials, kermes and the white oxide, may be administered, and better still in connection with the slight narcotics, such as aconite, belladonna in minute doses, or hyoscyamus.

Certain it is that emetics are excellent relievers of congestion of the bronchi. Vomiting, and even simple nausea, give rise to a deep perturbation in the local circulation, whence there follows, as proved by Pécholier, a considerable derivative action from the broncho-pulmonary apparatus. Nevertheless it is only in the acute cases, or those tenacious from the beginning, that this therapeutic measure need be resorted to.

The therapeutic indication yielded by the causes of congestive bronchitis, although less direct, is also very important. If for example, we are dealing with congestion in a tubercular patient, this diathesis by its presence is a further reason for action in order to forestall the degeneration of the products which congestion gives rise to around the tubercular foci; cutaneous revulsion will be chosen in preference to other procedures, and the antimonials, which are likely to irritate the intestines, and thus to open new routes of infection, or at least to aggravate the ulcerations which tuberculosis tends to produce of itself, should not be resorted to. Emollient fumigations, topical applications of the same nature, if needful, paintings with iodine, balsamic and sulphurous fumigations, such are the indicated therapeutic agents. In case of rheumatic patients, emollients in connection with local revulsives are to be especially advised, the counter-irritation being made on the skin and by the intestines. In a word, the etiological indications, although subservient to others, should be satisfied by the procedures required by the causal factor at work, without forgetting that, whatever the cause, it is in reality congestive bronchitis which is at the basis of the trouble.

I cannot conclude this lecture without referring particularly to the treatment called for in that tenacious form of bronchitis, which I have described: congestive bronchitis in the emphysematous, with angina and congestive irritation of the mucous membrane of the upper air passages, a variety of bronchitis which is rarely observed except in case of patients more or less rheumatic.

This affection is a very obstinate one, both as regards the phenomena accompanying the attacks and the recurrences. Revulsive agents act only as palliatives, and emollients are of no value. I have tested the administration of emetics, and the truth is that the diminution of visceral congestion, the tendency to perspiration, the general depression which accompany emetics, appear to be the most appropriate of all for overcoming the congestive erethism of the bronchi, so marked in the patients under consideration. But these unfortunate patients cannot be kept long enough in a state of permanent nausea, and the congestion returns and often in a more aggravated form when the medication is stopped. Therefore I have resorted to a medication which adds to

the decongestionating action of the nauseants a nutritive modification deeper and more capable of rendering permanent the acquired amelioration. I hence administer to these patients fractional, not large, doses of tartar emetic (.05 to .10 grammes) well diluted to facilitate tolerance. A teaspoonful of this mixture is given every hour, unless nausea prevents, and the administration is kept up for several days. Usually it has to be given for three days each week for several weeks in succession. To assure a cure I have sometimes deemed it useful to give a little arseniate of antimony, especially to patients in whom there exists disease of the heart as a complication of the bronchitis.

Typical Methods of Treatment Applicable to Congestive Bronchitis.

1. In case of congestive bronchitis in an infant cover the entire thorax with a linseed meal poultice, which is removed every six or eight hours. Every two hours administer one drop of tincture of aconite in syrup. Keep the bowels free by injections of oil or of glycerin.

2. In case of congestive bronchitis associated with beginning tuberculosis, order repeated sinapisms on the sides of the chest and on the back, alternated with revulsive frictions with ammoniacal liniment. In case of cough and insomnia give every hour a teaspoonful of a mixture containing tincture of aconite and syrup of poppies. Apply under the clavicle of the diseased side a blister, and in case of persistency in the symptoms touch the same locality with the actual cautery. The diet should be limited strictly for a time to soup and milk, all wine or exciting drink being prohibited. Confined, hot, dusty, atmosphere should be avoided. The temperature should be constant as well as the amount of moisture in the air.

3. In case of congestive bronchitis associated with disease and hypertrophy of the heart, rest in bed is indicated at the outset. Every three hours a teaspoonful of the following mixture should be administered:

℞ Extr. of Digitalis	.05	
Extr. of Conium	.10	
Powdered Ipecac	.15	
Oxymel. Scillæ	30	grammes.
Muc. Acac.	120	"

One to two pills like the following should be taken according to indication:

℞ Pulv. podophyl. 03
Extr. Bellad. 01
ℳ ft. pil.

In case of obstinate constipation order 15 grammes of sulphate of soda, frequently repeated; or else in a cup of cold milk,

℞ Pulv. scammon 50
Pulv. jalap 25
ℳ ft. chart. No. 1.
Milk diet strictly.

4. In case of congestive bronchitis associated with gout, apply here and there on the chest sinapisms. Give each morning a wafer containing powdered scilla (.10) and powdered phellandra (.25). Order with each meal seltzer water containing one-half a gramme of carbonate of lithia. Keep the bowels open by small doses of calomel (.20 to .25 grammes).

5. In case of congestive bronchitis in the emphysematous order (a) during the attacks small doses of tartar emetic repeated to nausea each morning for three consecutive days and continue for two to three weeks; rub the thorax repeatedly with a liniment composed of camphorated ammoniacal liniment and spirits of turpentine, or else with croton oil. When called for, administer a saline or drastic cathartic. Let the diet be of fruits, vegetables, milk, eggs and white meats. Vichy water is useful to counteract the local irritant effect of antimonials on the mucous membrane of the digestive tract. In the intervals (b) in the attacks give each morning, for five days in the week, 5 to 6 grammes of arseniate of antimony divided into milligrammes. Each night order a pill composed of extract of datura and powdered phellandra, āā .10 grammes. Tonic and non-irritating diet is to be ordered; sulphur fumigations each morning; the bowels to be kept free.

LECTURE VI.

CATARRHAL BRONCHITIS—ETIOLOGY AND SYMPTOMATOLOGY.

BEFORE beginning the study of catarrhal bronchitis, I deem it necessary to recall in a few words what is meant by the expression "catarrh," a term which is frequently erroneously used because those who apply it do not understand the exact definition.

Either the word is of significance at all, or else it refers to an elementary lesion which is none other than a hyperemia of the secreting surfaces. Unquestionably it is not easy to prove that this increase in secretion is produced without change in any of the products of secretion; but what seems well established is the fact that in the products of catarrhal secretion, even though there be increase in the watery elements, we find no increase in the solid elements which belong to the secretion, any more than noteworthy alteration in them.

Without doubt the three elements, congestion, catarrh, and inflammation may be conjoined, in so far as we rarely find any one alone. But if the hyperemia necessitates and causes a greater afflux of blood than is normal, it is because of its being a secondary phenomenon. Even as congestion may exist without hyperemia, even so may a catarrhal state be present without congestion. And further, even as there are special causes of congestion, even so are there special causes of catarrh.

I will not dwell on the necessity there is of differentiating catarrh and true inflammation. The distinction which I drew between congestive irritation and inflammatory irritation is perfectly applicable here. Catarrhal irritation causes a secretory flux, inflammatory irritation alters the products of secretion, multiplies the elements, changes more or less the same products either by plastic infiltration or by purulent degeneration.

We have here, you see, an elementary lesion very different from that of catarrh, and I dwell on the distinction no longer.

Now this catarrhal lesion may be met with on all secreting surfaces. The mucous membranes are the surfaces chiefly affected, they are the elective seat of catarrh. The mucous membrane affected by a catarrhal irritation secretes freely an aqueous liquid, or, as some express it, a serosity in which water predominates mixed with a certain amount of mucin.

All the mucous membranes may be the seat of this species of irritation, rather functional than nutritive, so to speak; but the mucous membrane of the bronchi is most frequently involved. Whence many writers consider the word catarrh a synonym for irritation of the bronchial mucous membrane.

This elementary morbid lesion is not the exclusive property of mucous membranes, however; it belongs as we have stated to all secreting surfaces. We may meet on the serous membranes an irritation more or less hyperemic whence result exudations which resemble dropsy by the watery composition of the products, and inflammation by the acuteness of their development. We are dealing here again with secretory irritation comparable in every respect to catarrh of the mucous membranes, and, besides, being often united with it in the same pathological group.

I have thought it necessary to recall briefly these principles which belong to general pathology, so that you will have no excuse for confounding, as many do under the pretext that they often occur together, phlegmasia, congestion and catarrh.

Catarrhal bronchitis may involve the large and the small bronchi, and even as in case of congestion, it is a very different affection according as it occurs in the one or the other. Again, like congestion it may be active or passive; and the distinction to be made between these two forms rests on the same basis both for congestion and for catarrh. Active catarrh is a hyperemia due to secretory irritation, resulting from an irritation acting on the secretory element itself; passive catarrh is a hyperemia following on stasis of blood or of serum, and this stasis, by increasing the tension in the neighborhood of the secretory surface, determines on this surface a species of exudation or of serous exosmosis; it is to this form in particular that the term *bronchorrhea* is applicable. The mechanism is, we thus see, very different in each instance.

In a word, there exist two forms of hyperemia of the bronchial mucous membrane—a first, active by direct secretory irritation; a second, passive, or increase of exosmosis by exaggerated vascular tension.

We will, as in case of congestive bronchitis, review the symptoms of catarrhal bronchitis, then its causes, varieties, evolution; and from this study we will draw the indications on which the diagnosis depends. Finally, we will deduce the practical consequences, by stating the therapeutics applicable to this form of bronchitis and the agents capable of satisfying the indications.

Symptoms of Catarrhal Bronchitis.—We will at the outset consider the subjective sensations, and these are practically *nil* or insignificant. At times the affection is complicated by certain neuralgic pains, either intercostal or elsewhere; but all pain may be lacking.

The functional troubles are very slight, or at least in accord with the extent and the degree of the catarrhal lesion. Almost nothing in the simplest and least extensive forms, the dyspnea may become intense in other forms; in these latter instances, indeed, the catarrh, having involved a considerable part of the bronchi, fills with its secretions the respiratory passages, and diminishes in so far the field of hematosis.

Cough is present, and is moist in accordance with the catarrhal secretion, the cough, however, is far less than in case of dry irritation. More frequent in the initial stage when the secretion has not answered the irritation which provokes it, the cough diminishes and becomes less painful afterwards, when it effects the expulsion of the moist products of bronchial secretion. This cough is full, sonorous, and marks by a species of bubbling the conflict between the air and the catarrh in the bronchi. It causes, besides, a sufficiently characteristic expectoration, salivary, fluid, transparent or foamy, opaline or citron yellow, rarely viscous and thick. It is ejected usually without much effort, often by a simple spitting; in such cases the patients may feel the liquid rise in the trachea and reach the pharynx. Exceptionally the cough is spasmodic, and this is dependent not on the hyper-secretion but on the nature of the irritant.

The physical signs yielded by auscultation are characteristic enough. These are moist râles which exist over the site of

the disease. When the irritation, purely catarrhal, produces an abundant secretion, fluid and readily expectorated, then large and fine râles are heard, moist, however, and sub-crepitant of varying calibre; while, on the other hand, if the products were viscous, the râles would be dry and vibrating.

You know that it is for this reason that the attempt has been made to make the two divisions of bronchitis with moist râles and bronchitis with vibrating râles; now of these two varieties, the latter is a mucous bronchitis to which the name of dry catarrh has been given, and it is generally only a congestive bronchitis, while the former belongs truly to catarrh and deserves the name of catarrhal bronchitis.

Percussion rarely yields any evidence in case of catarrhal bronchitis.

The symptoms from the lungs themselves are negative; that is to say, the pulmonary parenchyma is not altered, and there is neither dullness on percussion nor souffle on auscultation.

The mucous membranes of the upper air passages, however, furnish evidence of the part they play in the affection, and especially by coryza and pharyngitis. These irritations are themselves catarrhal and manifest this character by a certain congestion of the mucous surface and by a more abundant secretion; from the side of the larynx there is some change in the voice.

The general phenomena vary according to the cause and also according to the extent and the depth of the catarrhal bronchitis. In the simplest forms of the disease they are more or less lacking; and this constitutes one of the varieties of what is commonly called a cold. Catarrhal bronchitis is almost always accompanied by a febrile reaction, often light enough not to attract notice, and then again characterized by chills, high temperature, abundant perspiration. At the same time there prevails a low state of health, at first constipation predominating and then diarrhea. Hence good observers have thought that catarrh had a tendency to progress from above downwards, and beginning in the respiratory tract to finish in the digestive.

LECTURE VII.

CATARRHAL BRONCHITIS—CAUSES AND VARIETIES.

1. THE most simple form of catarrhal bronchitis is that to which the term *rheum* (ρευμα, a running) has been applied. It has an initial stage of greater or less duration, often totally lacking, and characterized by a dry (perhaps congestive) irritation of the bronchial mucous membrane. In any event this initial stage is accompanied by slight or ephemeral fever followed by perspiration. The expectoration, at first *nil*, is soon established, and is abundant, more or less limpid, and is accompanied by an easy cough.

This affection, however slight, has a certain tendency to persist. The fact alone that the local character of this disease consists in a simple exaggeration of a normal secretory function seems to render return to the normal use of this function more difficult. In fact, the slightest occasional cause is sufficient to renew this simple functional exaggeration, which constitutes the element catarrh; and where the subject in whom this affection recurs is at the same time under the influence of a constitutional diathesis, this in turn contributes to feed or favor recurrence of a catarrh once contracted, so that the duration becomes, as it were, interminable.

2. A further variety differs only from the preceding in that the elevation of temperature is a trifle more marked, and the duration longer, at least one week. The patient must cease from work, the fever is continuous with evening exacerbation. The bronchial localization is more pronounced, more general and deeper, as witness the râles heard on auscultation.

The fever is entirely characterized by the remission, the defervescence and by what are called the critical eliminations. From the fifth to the ninth day these crises recur either by the skin (diaphoresis), or by the urine (diuresis). Towards the seventh day defervescence occurs. In a word, this form is not different from the preceding except that the local and the

general manifestations are more severe and the duration of the fever is more marked.

3. *Influenza* is nothing more than a catarrhal fever, epidemic in character; it is a pyretic affection, epidemic and contagious—perhaps virulent—probably bacillary.

It is a febrile affection, I have stated, and a fever which, in its most perfect evolution, lasts for three weeks. Preferably it pronounces itself as a catarrh of the respiratory tract, although occasionally it affects the digestive mucous membrane.

The symptoms are: at the outset general malaise with depression; next irregular chills and a more or less remittent fever, usually with evening exacerbation. Often these exacerbations are marked enough and are accompanied by a constitutional depression deep enough to give the appearance of a grave pyrexia. Nevertheless the affection does not resemble typhoid; it lacks the depression and cerebral torpor characteristic of typhoid patients.

The principal localization of this pyrexia is on the bronchial mucous membrane, where it assumes the catarrhal form. On auscultation we hear mucous râles disseminated in the bronchi, large and fine mingled, extended over a variable portion of the thorax and often over the entire bronchial system.

Almost always the catarrh invades concomitantly the other mucous membranes. From the outset the tongue is coated; soon it clears and the intestine becomes the seat of a true catarrhal diarrhea. The pulse, which is frequent, remains variable. Secondary congestion occurs in different organs, sometimes, indeed, true hemorrhages. The lungs, liver, spleen, are the seat.

Finally, from the side of the nervous system, there exists a state of asthenia, rather than of true adynamia. In typhoid fever, the nervous condition seems to result from the poisoning which affects the subject from without or from the digestive apparatus; in influenza this poisoning exists as well, but its source appears to be rather the nutritive alteration of the muscles. The ptomaines of typhoid are further of a different and more septic nature. Whatever the case the asthenia differs notably from the adynamia of typhoid fever.

Delirium is often present in the aggravated forms of the affection, and this delirium is more active than that of typhoid; occasionally there are marked neuralgic pains and even con-

vulsive phenomena. The perspiration is always more or less abundant, and determines often eruptions.

Complications from the side of the respiratory tract are not rare; the various kinds of bronchitis, including the capillary, broncho-pneumonia and even true pneumonia occur; and these affections are then exceptionally grave, not alone because of the ease with which they extend and become generalized—not alone, further, by their tendency to become inflammatory and even septic—but also because of the general state of critical asthenia which is always an accompaniment.

I do not intend to sketch the whole history of the affection, but only by recalling its general traits to show you the part which catarrhal bronchitis plays. I want also to impress upon you the other pyretic affections during the course of which catarrhal bronchitis may occur and with which it should not be confounded. I have spoken of typhoid fever in the course of which we meet congestive bronchitis commonly, and the same holds true of measles.

4. *Catarrhal bronchitis propagated from the mucous membrane of the upper air passages.*—Coryza, as we have stated, often accompanies catarrhal bronchitis; frequently indeed it is the starting point, so much so that the opinion has been held that bronchitis is an extension of the catarrhal irritation of the mucous membrane of the nose to that of the bronchi. Certain kinds of coryza appear to have a special aptitude for such a propagation; such is catarrhal spasmodic coryza known as hay asthma; the catarrhal laryngitis of false croup also is often enough complicated with catarrhal bronchitis.

5. *Catarrhal bronchitis of pulmonary origin.*—*a.* Catarrhal bronchitis is often associated with the various neuroses of the respiratory apparatus. The catarrhal irritation almost always results in a measure from the attacks of asthma and seems to terminate them; at the end of the dry and spasmodic phase secretion is produced in the bronchi, and manifests itself by moist râles and by expectoration. Now the catarrhal irritation thus established may become more or less permanent in the asthmatic, and even though it be not permanent it seems to recur with greater facility with each attack.

The same holds true for whooping cough. At the end of the attacks there results a catarrhal expectoration and this

may amount to a true catarrhal bronchitis, and so remain permanently.

b. In case of *emphysema* bronchial congestion is more frequent than catarrh, at least in the acute stage; nevertheless the repetition of the congestive attacks leaves behind a chronic catarrh.

c. Pneumonia has a close affinity with catarrhal bronchitis. There is even a form of pneumonia which has secured the name of *catarrhal pneumonia,* owing to the chief *rôle* which the catarrhal element plays in its evolution. In a paper which I published on this subject (*Archives de Médecine,* 1862) I endeavored to sketch the characteristics of this species of leucophlegmasia, which is not a pneumonia complicated by bronchial catarrh, as many have thought, but a species of hyperemia at the same time catarrhal in the bronchi and congestive in the parenchyma itself of the lung.

Further, around each true pneumonic focus there is a zone of active serous infiltration, which is only congestion, the mechanism of which is hyperemia, and this is of the nature of catarrh. Finally, above all, after hepatization, the lungs are the seat of a hyperemia more or less congestive, more or less secretory or derivative; more congestive before hepatization, it constitutes the first period of the pneumonia; more derivative afterwards, it is the sign of resolution.

Often at the autopsy, you have seen these peri-inflammatory or post-inflammatory zones, which on section give issue to an abundant serosity scarcely mixed with blood; and this proves how the alveoli and the intercellular spaces of the pulmonary parenchyma are invaded by the serum. Now this same process goes on at the same time at the surface of the bronchial mucous membrane around the inflamed pulmonary lobule. Often even the congestion extends beyond the lobule to the pleura and then occur those slight attacks of pleurisy which you see so often accompany pneumonia during the period of defervescence.

From the side of the bronchi there exist râles and expectoration. Sometimes I have found in the expectorated matter coagulable albumin, and this is seen in particular in the catarrhal expectoration which follows so abundantly on thoracentesis.

d. In *pulmonary edema* two things may happen: either it

is one of those acute edemas in which the secretory irritation plays the chief *rôle,* active edema *par excellence,* and in this instance the secretory irritation may extend from the pulmonary parenchyma to the surface of the mucous membrane; the resulting catarrh is acute, more or less intense and extensive; it may even simulate suffocative catarrh; or else, in other instances, the edema is passive, produced, for example, by a mechanical obstacle in the circulation, hypostatic edema as it is called, and the resulting catarrh at the surface of the bronchi is both passive and mechanical. All the causes of slow and progressive dropsy may at any time be accompanied by this torpid form of catarrhal bronchitis.

e. In case of diphtheria we often observe a prodromal period of catarrhal bronchitis; above all in the infant I have often seen this catarrh prelude and precede by some days the appearance of the false membranes.

Catarrhal hyperemia is also met with in diphtheria at the time when the mucous membrane prepares to eliminate and to throw off the false membranes. It is understood, of course, that the catarrhal secretion plays a part in the expulsion of the epithelial layers infected by the diphtheritic products even as it assists in the elimination of the slimy products of the digestive mucous membrane. When diphtheria presents the characteristics of catarrhal bronchitis, when we find moist râles and expectoration, then there is ground for giving a relatively good prognosis.

f. Nothing is more common, we have stated, than congestive bronchitis around tubercular foci; catarrhal bronchitis, although less frequent, is met with during the course of *tuberculosis.* It arises, at the outset, around the foci, and it is certain that, even as in case of congestion, catarrhal bronchitis often precedes the eruption of the tubercle.

It is a fact that the epithelial desquamation which accompanies the catarrhal process, in case the products are retained in the air passages, leads to a favorable soil for the reception and the multiplication of bacilli, and it is hence apparent that popular opinion in regard to a neglected cold is not without foundation. Finally, catarrhal bronchitis always accompanies to a degree the transformation or the softening of the tubercle, and it is then perhaps due to the passage of the eliminated

products over the bronchial mucous membrane, and to the direct irritation of this surface.

Whatever, still, be the influence of the tubercle in increasing the catarrhal irritation in the bronchi of the phthisical, this seems to be due less to the presence of the tubercle itself than on the presence of a diathesis. Whence it is why catarrhal bronchitis is found by preference in scrofulous individuals and in the torpid varieties of phthisis; while in the active forms, in case of rheumatics in particular, the bronchial attacks are especially congestive, even as they recur more frequently and tend less to become chronic.

g. I have already noted in connection with pneumonia the relation existing between *pleurisy* and catarrhal bronchitis. I have noted this even in a case of dry pleurisy where the patient, a man, without having presented any evidence of exudation, was relieved greatly of a basal pleurisy after having expectorated during an entire night an abundant serous secretion, that is to say, catarrhal.

h. Ganglionic phthisis, or tuberculosis of the peri-bronchial ganglia, is readily complicated by catarrhal bronchitis. Often it results from reflex irritation; at times it depends on vascular compression, and again on irritation of the vaso-motor or the mediastinal nerves.

Finally, all the organic affections of the lung, and, we may add, of the entire thoracic cavity, are capable of causing catarrhal irritation of the bronchial mucous membrane, either through vaso-motor disturbance, vascular compression, or more or less direct secretory irritation.

6. Catarrhal bronchitis is frequently associated with *cardiac disease.* Those patients are the more likely to become so affected than others owing to the alteration in nutrition of the bronchial mucous membrane which results from imperfect circulation. Whence the frequency of colds and their gravity and persistence in case of cardiac disease.

Patients with cardiac disease often are affected concomitantly with a passive bronchial catarrh; this is connected with the stasis of blood which we note in an early period of cardiac disease, especially the mitral and tricuspid variety; if to this catarrh a factor of secretory irritation is added, catarrhal bronchitis becomes established. Finally, the low state of health into which these patients eventually lapse, in particu-

lar those subject to albuminuria, leads to edema, at the outset acute, and for the same reason to catarrhal bronchitis.

Dyspeptics often also present catarrhal bronchitis, and similarly sufferers from affections of the *liver.* In case of *nephritis* the iritation of the bronchi is caused by the abnormal elimination of urea and its derivatives, and from this irritation there readily results catarrhal bronchitis. Often accompanying nephritis we witness alarming attacks of suffocative catarrh. I will cite an interesting case soon when speaking of scarlatina.

Catarrhal bronchitis is also a frequent accompaniment of the majority of the morbid *cutaneous* manifestations, whether because of the physiological relations existing between the skin and the bronchi or for other reason we cannot state.

It is in the course of the *eruptive fevers* that we see this most frequently; indeed catarrhal bronchitis is an integral part of one of them—*measles.* Without question the bronchitis of measles is not always catarrhal; it may become congestive, and even a graver form still; but it is certain that it begins by moist secretion from the upper air passages, no matter what the congestion which is superadded.

This bronchitis has also a great tendency to become generalized and to extend in depth even so far as to become a catarrhal pneumonia. You know, further, how readily it may alter into tubercular catarrhal bronchitis, especially if the subject is at all so predisposed.

In case of simple *scarlet fever* catarrh is infrequent; but the renal complications of scarlatina are often associated with congestive or catarrhal bronchitis, suffocative and subacute.

I would mention in this connection the case of an infant I was attending some time ago during the course of scarlet fever. Defervescence had occurred, and everything was progressing favorably except for a slight quantity of albumin in the urine. This fact, however, caused me to give a guarded prognosis. A few days afterwards the child was seized by a suffocative catarrh which killed it in a few hours.

I will not dwell particularly on bronchitis accompanying *typhoid fever,* since it rather assumes the congestive than the catarrhal form. Two causes in especial tend to provoke the bronchitis: the passive stasis of the pulmonary circulation

and the greater or less change in the blood which is peculiar to the disease.

I will say nothing more about the *eliminative* form of bronchitis; after what I said under the subject of congestive bronchitis it is sufficient to recall the fact that certain medicinal agents, eliminated by the bronchial mucous membrane, cause a greater or less irritation. Since this action is often utilized therapeutically, I will return to the subject when speaking of treatment. Remember only the principal agents which are likely to thus produce catarrhal bronchitis: certain salts of potassium, especially the iodides, all the balsams and the sulphurs.

I have already stated that there are certain catarrhs strictly diathesic; this cannot be denied in connection with scrofula, and we see many infants in whom other manifestations of scrofula, such as impetigo, coryzas, anginas, alternate with more or less torpid attacks of catarrhal bronchitis.

Rheumatics are not protected. Although in rheumatism and gout we notice rather congestive than catarrhal manifestations, the latter are also met with especially before the diathesis has acquired or after it has lost its acuteness or at the time when the patient's constitution begins to weaken.

7. Age, sex, etc., need hardly be considered in the etiology of catarrhal bronchitis. As for the influence of season I could not add to your knowledge, for I have often laid stress on it.

LECTURE VIII.

CATARRHAL BRONCHITIS—CLINICAL HISTORY AND DIAGNOSIS.

WHAT is the evolution of catarrhal bronchitis? It does not always bear the same characteristics; active or passive, acute or chronic, it may appear under very different guises. Its simplest form is that of a slight rheum, lasting for a few days, accompanied by scarcely any fever and deserving truly the name *ephemeral catarrhal fever*. The fever may be more pronounced, lasting for seven days to three weeks and over, and yet the characteristics may not be specially altered.

The pyretic, serious form of catarrhal bronchitis is a better defined condition both etiologically and symptomatically. It consists in: 1. a period of invasion, characterized by an acute bronchial catarrh with a general state of profound depression. 2. A period of febrile reaction more marked but less adynamic than typhoid fever in general, coinciding with more pronounced phenomena of bronchial irritation, and more catarrhal as well, as is proved by the expectoration. 3. A final stage marked by excessive perspiration, the defervescence going on by degrees up to complete recovery.

This typical course is often varied by numerous irregularities, which may modify considerably one or another of these periods. Thus the type may change from remittent to intermittent. This is especially true of the third stage; often indeed, as has been noted by Dr. Carrière, the intermittence is such that it may be looked upon as a special morbid entity calling for quinine, and this too aside from any paludal influence. It goes without saying that this type is peculiarly likely to be met with in subjects who have previously had intermittent fever.

I will not enter at length upon the history of this affection, since it is well treated of in our works on pathology. I only desire to add that it is often serious and sometimes fatal;

usually, except in the very young or old, it is more distressing than dangerous. In infancy, the patients are likely to die of broncho-pneumonia, and this sometimes happens in adults; in old age it is particularly asthenia which is met with and which is to be feared. In such instances it may be complicated by a true typhoid condition. It is finally an affection dangerous to those already subject to broncho-pulmonary affections, to the asthmatics, for instance, and to the tubercular, in whom it leads to the formation of new foci and especially hastens the evolution of the old.

Whilst it is common enough to see congestive bronchitis change into catarrhal, it is quite rare that we find catarrhal altering into congestive bronchitis. This is possible, however, and the transformation rarely stops at the congestive form but reaches often at once the inflammatory. This transformation may be the result of imprudence on the part of the patient or even to premature attempts at substitutive therapeusis.

It sometimes happens that catarrhal bronchitis, without being either epidemic or contagious, becomes remittent or intermittent, and this too not only as regards its general febrile phenomena but also as regards the local lesion. Even as in intermittent fever, variations in pyrexia may exaggerate one or another of the periods, so in some sufferers from catarrh the severity of the chill, in others the acuteness of the fever, in others the abundance of the perspiration, may suggest a pernicious element.

The suffocative form of catarrhal bronchitis has always received due notice from writers, but it has always been more or less confounded with the inflammatory or congestive forms of this disease. The best known type is that seen in cases of acute anasarca.

There is at present in our wards a patient with catarrhal bronchitis, who expectorates habitually a great quantity of clear fluid. For some days this expectoration has been extreme, and asphyctic phenomena show themselves, whilst the air penetrates with difficulty into the thorax. The bronchitis became suffocative without our being able to determine the cause of the increase in his symptoms. It may be due to a vaso-motor irritation, such as compression of the pneumogastric, or else to a direct vascular change

Whatever our doubt as to the cause of such phenomena, I want to remind you that, the fact of excessive secretory irritation once well established, the therapeutic indication is clear; you will understand, hence, how important it is to determine the exact form, whether congestive, inflammatory or catarrhal, of such phenomena. Besides, this intra-bronchial serous hyperemia may vary a trifle in its acuteness and its morbid physiognomy, according as it is a question purely of a dropsical hyperemia, or, on the other hand, of a more or less active fluxion. This, in chronic dropsy, is pulmonary edema itself. A serous hyperemia, slow and chronic, occurs on the bronchial mucous membrane; and in acute anasarca, the form in particular which is seen in albuminuria and in convalescence from scarlet fever, there is shortly produced a serous secretion in the respiratory track, sometimes even albuminous, and the patients die speedily as though from an inundation of the bronchial tubes.

The *chronic* form of catarrhal bronchitis is generally diathesic. You have seen how often the scrofulous readily become subject to chronic catarrhal bronchitis; therefore when we meet with a persistent bronchial catarrh it is always well to hunt for a scrofulous origin.

Scrofula, however, is not the sole diathesis which disposes to the chronic form of catarrhal irritation of the bronchi. Rheumatism is certainly less frequently a cause; its preferable manifestation is rather acute catarrh or the congestive form. There is, however, a further pathological relation which it is necessary to bear in mind, and this that existing between catarrhal irritation and certain cutaneous manifestations: thus bronchial catarrh is frequently met with in connection with eczema, prurigo, lichen; and this same relation may be noted between cutaneous affections and spasmodic bronchitis.

Diagnosis of Catarrhal Bronchitis.

When we began the study of this affection I recalled the characters which separate the catarrhal from the congestive and from the inflammatory element, and we saw that the expectoration constituted one of the principal points of distinction.

None or scarcely any at all, and then viscous and adherent

in congestive bronchitis, the expectoration is purulent or mucopurulent and varying in amount in inflammatory bronchitis. In catarrhal bronchitis the expectoration is profuse, fluid, cloudy or clear, like the saliva.

By auscultation we may establish the seat, the extent and above all the depth of the catarrhal lesion; the febrile state, its acuteness and above all the type it affects, measure the gravity of the affection and indicate often the etiological conditions on which it depends. The intensity of the general reactionary phenomena, the asthenia which is marked from the outset, the easy generalization of the local phenomena will distinguish the epidemic form; the absence of true adynamia, the slight importance of the gastro-intestinal phenomena, the predominance of the bronchial signs, these are the points which will differentiate from typhoid fever.

It is often a delicate point to determine at the outset catarrhal bronchitis from measles. In the beginning measles is announced by a catarrhal bronchitis, and this may last from three to five days; and, not very rarely, towards the third day, the temperature falls notably to rise again at the time of eruption. Here is a diagnostic difficulty which we should always bear in mind. There are, however, shades of difference between these two morbid states; rubeolic bronchitis is as much congestive as catarrhal; this characteristic is determined by the redness which from the beginning it impresses on the pharynx, by the acuteness of the coryza and the conjunctiva.

As for the diagnosis of the causes of catarrhal bronchitis, we have seen that they are to be sought in the condition of the mucous membrane of the upper air passages, in the state of the lungs, in diphtheria or tuberculosis, for instance, or else in the viscera, such as the heart, liver, kidney; the examination of these organs will soon prove the share they have in the evolution of the disease. The same holds true of the eruptive fevers, intoxication and diatheses, causes on which I need not longer dwell.

LECTURE IX.

TREATMENT OF CATARRHAL BRONCHITIS.

WHAT elements of catarrhal bronchitis ought our therapeusis to be aimed at? You know that in the determination of the indication, the category of elements which it is essential to bear in mind is that which determines the form of the disease, formerly called the physiological element, as being the one which we can the most effectively attack.

Here this element is easy to determine with exactness: catarrhal bronchitis belongs to the class of secretory alterations; it is above all a quantitative alteration; in a word, it is a diacrisis which may be either acute or chronic.

It is easy hence to understand that the indication to fulfill is a triple one:

1. Diminish the excessive secretion; 2. Eject the secreted products, or at least favor their expulsion; 3. Modify the secretion itself.

a. There are a number of means at our disposal whereby we may diminish bronchial secretion—at the outset the decongestive agents.

Although catarrhal irritation is not necessarily connected with congestive irritation, it is established that the secretory product of catarrh is derived from the blood which irrigates the secretory surfaces; consequently, whatever abates the circulation of the bronchi will diminish at the same time the source of the bronchial secretions.

There is still another reason for attacking the catarrhal irritation by means of decongestive agents, and this is that however catarrhal it be the irritation induces and leaves behind it a blood stasis which favors in turn the production of catarrh. Whence the double utility of fighting indirectly the catarrhal element by attacking the congestion.

We saw in one of the preceding lectures how this is possible, either by means of emollients, or by nauseants, or by re-

vulsives, which are so efficacious in case of congestive bronchitis. Although emollients seem to be of less efficacy against catarrhal than they are against congestive irritation, it is nevertheless well to use them, and sometimes they will fulfill the indication. After having removed all the conditions capable of provoking a new irritation, even as I have shown in the lecture on the treatment of congestive bronchitis, we may in the simple forms prescribe emollient drinks.

Some writers, the Germans in particular, have deemed it necessary to do as much as possible without liquids both in medication and in diet, in case of catarrhal affections, the aim being to diminish absorption and consequently to retain in the blood itself the source whence the secretions obtain their materials. This theoretical view is not without usefulness. Still it could only be exact if the secretory action was absolutely dependent on both the quantity and the quality of the blood in particular; now we know that this is not the fact, and in true catarrhal irritation we are rather dealing with the functional irritation of the secretory agents, glands and surfaces, than with the materials brought to them, either indirectly by the blood or more directly still by the routes of absorption.

We may give, then, in catarrhal bronchitis, emollient drinks, but fumigations are preferable, the topical action of which is most marked. Fumigations by infusions of emollients are very useful. Fomentations and cataplasms on the thorax, so useful in case of congestive bronchitis, are of no utility in case of catarrhal bronchitis.

Catarrhal irritation attacks secreting surfaces and scarcely affects parenchymatous tissues; hence the use of deeply penetrating revulsives is not indicated as in case of congestive bronchitis. This is the reason why we obtain the best results from diaphoretics. With this end in view Laennec gave punch to cases of recent bronchitis, hoping thus to abort the affection.

Superficial revulsives, rapid and transient in action, may also be used. Cutaneous revulsives, such as sinapisms, irritant frictions, alcoholic or ammoniacal, if need be a few blisters, especially those which are left on only a few hours and are then replaced by poultices. This practice is all the more useful here, because the blister, in order to be efficacious, should cover

a large surface, and you know how important it is not to leave large blisters in contact with the skin to avoid cantharidal poisoning and vesical troubles to which they may give rise.

Intestinal revulsives, in particular those which act on the mucous membrane and on the glands of the intestines, are doubly useful. Their action on the mucous membrane of the intestine is derivative from that of the bronchi; besides the loss of fluid which they provoke tends to diminish the exaggerated secretion. With this end in view the dialytic purgatives may be used to advantage; the neutral salts, sulphate of soda and magnesium, the alkaline citrates, tamarinds, pulsatilla, and, if need be, calomel.

When it is necessary to act quickly and energetically, as in the serious forms, and especially in the suffocative, the drastics are indicated; aloes, jalap and scammony, croton oil. This drastic medication would also be useful where the secretion, without being excessive, is obstinate in duration, and where we must strike hard to overcome a bad habit.

As for the nauseants it is only the serious forms of catarrhal bronchitis which require them, at least as decongestive agents, for we will soon see that they play a very important *rôle* as expectorants.

We may further diminish the secretion of the bronchi by attacking the nervous excitation which accompanies it and often provokes it. All the depressants of nervous action are here indicated. But, like the decongestives, they exert on the secretion only an indirect and a little uncertain effect. Aconite, hyoscyamus, conium, are often used, as well as the weaker preparations of opium, etc. These agents, as also arsenical preparations, will be studied particularly under the subject of spasmodic bronchitis.

The aromatic umbelliferæ constitute with the antispasmodics the transition between the agents which moderate innervation and the derivatives. They can only be used when the bronchial discharge, having lost its acuteness, tends to become chronic.

We are thus naturally brought to speak of the agents which directly diminish secretion. In the first rank come opium and its compounds—laudanum and morphia. In this connection, however, opium is much inferior to belladonna and all the solanaceæ, hyoscyamus, conium, and especially datura;

their alkaloids, atropine, hyoscyamine, cicutine and daturine seem to be less efficacious.

These different drugs are best administered in small doses, frequently repeated in solution, pill or powder. The extracts may be given in doses of five to twenty centigrammes, divided into pills of two to four to five centigrammes. Ipecac in very small dose may diminish bronchial secretion; the nauseants in general, especially antimonials, in small dose, have the same effect.

These various agents have in particular the purpose of diminishing the bronchial secretion; some are opposed to excretion, whilst others favor it.

b. The second capital indication consists in favoring the expulsion of the secreted products. The therapeutic agents which fulfill this aim are known by the characteristic name of *expectorants.*

The great majority of these agents are above all capable of modifying the products of secretion and of rendering them more fluid, and also of provoking contractions either of the bronchial muscles or of the adventitious expiratory agents, the conjunction of which is necessary to effect expectoration.

In the first rank we may mention acidulated drinks, the nitrates of potass and soda, polygala, squills, gum ammoniac, even jaborandi; and in the second rank, the nauseants, and above all the emetics, ipecac in small repeated or in one large dose, antimonials in general, kermes, arseniate of antimony and white oxide.

Polygala is given especially in infusion; squills in powder of from five to fifty centigrammes or in the shape of oxymel scillæ (ten to thirty grammes); gum ammoniac, one to five grammes, in emulsion, or mixed with the oxymel to make a pill; ipecac one to two grammes, in emetic dose, or twenty centigrammes, repeated; kermes in tablets, and arseniate of antimony, two milligrammes to one centigramme.

Excitant fumigations of chlorhydrate of ammonium and of carbonate of ammonium may be of great utility, both in awakening the sensibility of the mucous membrane and in provoking the expectoration of the products of bronchial secretion.

Jaborandi should be reserved for those cases where there is much fluid in the bronchi. It may be given in infusion; the injection of pilocarpine is still more rapid and more certain.

c. The third indication consists in so modifying the mucous membrane that it shall cease to pour out in excess liquid into the bronchi.

I have told you how tenacious and likely to recur is catarrhal irritation of the mucous membrane; and the bronchial mucous membrane constitutes no exception. So true is this, that the word catarrh means to the majority of people something chronic. This is the reason why we must use energetic modifiers, such as the balsams and the sulphurs, or alterative substitutives such as the antimonials.

These latter agents, to range them in the order of therapeutic activity, comprise the white oxide of antimony, turpeth mineral, and tartar emetic. The white antimony may be given in the dose of two to four grammes; kermes in the dose of thirty to forty centigrammes; and tartar emetic in from five to twenty-five centigrames, repeated as is indicated in the third lecture; the yellow sulphate of antimony in the dose of thirty to forty centigrammes.

There are many balsams which may be used to advantage in catarrhal bronchitis. I warn again, however, that it is well to be careful at the outset with this medication. The balsams always excite to a degree the mucous membrane of the bronchi, and thus the affection might be aggravated, if they were employed too soon or too strong. They may, as I have stated, transform a catarrhal into a congestive bronchitis, and this is far from being an advantage; still, when used opportunely and in moderation, they may check the hyperemia and restore the normal.

This medication should only be used in case of the torpid and chronic forms. The agent most usually employed is turpentine, and this is generally only applicable to the above forms; it is best to begin with resin capsules, and only later to order capsules of the essence of turpentine. Myrrh, benzoin and styrax are rarely used; balsam of Peru and of Tolu is often used. Tolu particularly is most useful and very agreeable. The essence of citron has also been given. The preparations of camphor are but little ordered. Formerly gum ammoniac was much used; and energetic agents are found in the ammoniacal salts. The carbonate and the benzoate of ammonium may be given in the dose of .25 to 2 grammes. The action of creosote is similar to these agents.

After the balsams come the sulphurs. The usual method of administering them is in natural sulphur waters, which are ordered in the morning fasting, pure or, better still, mixed with a quarter or a half the quantity of hot milk. The best waters are, in order, that of Cauterets; of Enghien; of Saint Honoré; of Labassère; of Eaux-Bonnes. Further still, artificial sulphur waters may be ordered, and these waters may be used as gargles, fumigations or pulverizations.

LECTURE X.

INFLAMMATORY BRONCHITIS—VARIETIES AND SYMPTOMS.

If we were writing a monograph on bronchitis, then the study of the inflammatory form ought to detain us as much as the other forms together; but I only aim here at pointing out to you the boundaries which separate this form from the others, and above all to show you in what way they are related, in order that you may be enabled to differentiate the pathology of these various morbid forms. For further information I must refer you to the classical works at your disposal and especially to the articles which have recently appeared in our cyclopedias.

The care which I took in the preceding lectures to differentiate catarrhal from both congestive and inflammatory bronchitis renders it unnecessary for me to insist further on this point. Let me recall, however, before proceeding further, that the inflammatory form unites in itself both congestion and catarrh with something else superadded. Inflammation resembles congestion in so far as there exists sanguineous stasis and plastic exudation, and as for the catarrhal process it differs from it in that there results a fibrinous exudate and by the addition of neocytes incapable of life except amidst degeneration—that is to say, there is superadded pus. There is present, then, in inflammation more and something different from what both congestion and catarrh entail, and this is the reason why I endeavor to uphold the separation of these diverse elements, formerly differentiated clinically, and which physiological analysis allows us to-day to distinguish more accurately than was formerly the case.

Thus differentiated from congestive and from catarrhal bronchitis, inflammatory bronchitis, strictly so called, is of far less frequent occurrence than was formerly held to be the case. It is, nevertheless, worthy of holding a high rank in this study and comprises numerous varieties: at the outset

catarrh of the large bronchi, and catarrh of the fine. This latter variety, even as was the case in the preceding forms, both the catarrhal and the congestive, plays a large *rôle* in inflammatory bronchitis; and on this point I will have occasion to insist.

Inflammatory bronchitis may be acute or chronic, like all inflammations. As for the varieties called dry bronchitis and moist bronchitis, we need not further occupy ourselves, the first being nothing more than congestive bronchitis, and the second only catarrhal bronchitis.

These matters being understood, I shall proceed to make as careful a study of inflammatory bronchitis as I have of the other varieties, bearing always in mind the methodical analysis of the elements of the disease and the practical applications of which you should always be cognizant and which are my chief aim.

Symptoms of inflammatory bronchitis.—The subjective phenomena belonging to inflammatory bronchitis are a feeling of retro-sternal pressure which sometimes becomes a sharp pain with a sensation of more or less burning. It is noteworthy that this sensation is less frequently marked than it is in case of congestive bronchitis; at any rate, when it is acute, it is less persistent; in a word, it seems to be more dependent on the acuteness of the congestion than on the inflammatory process.

The objective signs are functional derangements which are in relation with the extent of the lesion along the bronchial tree. These are cough and dyspnea, and these scarcely differ at all from what we have noted under the other forms of bronchitis.

The expectoration, on the contrary, is very significant; it is scanty at the outset, but it soon becomes more abundant than in simple congestion; furthermore, it may for a time simulate the characteristics of the catarrhal form, which you are familiar with, since I have so recently described it and you have seen it so often at the bedside of patients; still, more viscous, the expectoration soon takes the purely muco-purulent character—that is to say, it is greenish, adheres to the vessel in which it is expectorated and falls into a more or less homogeneous mass.

The physical signs are less characteristic but not without

value. Percussion is negative. On auscultation râles of various calibres are heard at the outset; soon sub-crepitant râles, of a calibre proportionate to the size of the bronchi in which the inflammation is seated, are heard, and finally they are the only ones detected. Loud, moist, and bursting are these râles when in the large bronchi, and in the fine bronchi they are small in calibre if the air penetrates into the smaller bronchi, and they become less and finer as the bronchi are rendered impermeable by the inflammatory process.

The phenomena from the side of the upper air passages are, in general, less marked; the deep inflammatory form has less tendency than the preceding to affect the contiguous mucous membranes. The same does not hold true as regards propagation to the pulmonary parenchyma; true inflammatory bronchitis is frequently complicated with broncho-pneumonia.

Fever is constant in inflammatory bronchitis; it is present from the start, is often quite acute at the time when the lesion passes from the period of dryness to the period of secretion, and generally at once begins to decline. This also holds true in proportion to the extent, and especially to the depth of the inflammatory process. The temperature reaches its limit when the sanguineous stasis begins to complicate the anhematosis due to the bronchitis and then there is tendency to asphyxia.

The symptomatology of inflammatory bronchitis may then be resumed into the following three elements: 1. First, the inflammatory secretion which is muco-purulent and sharply differentiates this form from the two preceding; 2. The more or less serious pyretic state, which is always marked, at least at the beginning of the affection; 3. The always pronounced dyspnea, the greater the more extensive the affection, even to the degree of asphyxia.

Causes and varieties of inflammatory bronchitis.—1. Simple bronchitis, the result of cold, in the form I have just described. It begins like the varieties we have already studied, only the febrile movement is more accentuated. The fever is continuous, and the phenomena vary according as we are dealing with a bronchitis of the large, the mean, or the capillary bronchi.

To this variety is closely allied bronchitis *a pulveribus*, which results from the inhalation of an atmosphere charged

with dust, and that which follows the inhalation of irritant gases.

2. The most important variety, the one on which we must dwell in particular, is *capillary bronchitis*—that is to say, an inflammation which has reached a greater or less number of the bronchioles, those which approach the pulmonary vesicle and which differ from it only in that the epithelium is distinct and the vascularization is very different, even as Robin has established, as I recalled to you at the beginning of these lectures.

Capillary bronchitis is an inflammatory affection of the bronchial mucous membrane, the habitual course of which is from the large bronchi towards their termination; therefore it has less tendency to become localized than pneumonia for example, which seizes at once upon a lobule of the pulmonary parenchyma.

The progress of capillary bronchitis is closely related to that of broncho-pneumonia, and the frequency with which these two lesions are associated allows us to judge of the relation existing between them. Still, if you read the article which M. Blachez devoted to the study of this question in the *Dictionnaire de médecine pratique,* and that which my teacher, Dr. Roger, whose ability is well known, has written, you will be convinced that the two diseases should be held as distinct, both anatomically and clinically.

Capillary bronchitis is characterized by its inflammatory form, by its seat in the deeper bronchi, and the ease with which it spreads over the entire bronchial tree, by its etiological conditions (for it is usually a secondary affection), by the group of symptoms which accompany it, such as ulcerations of the mucous membrane, atelectasis by the blocking of certain air channels, and finally by the characteristic suffocation which is the chief accompaniment of the high fever.

Whether a special microbe be found in the affected bronchi or not, these lesions may still lay claim to an inflammatory character, and, what is specially characteristic of this affection, aside from its cause, is the fact that it is extensive, general, and is readily complicated by pulmonary congestion, emphysema, and broncho pneumonia. For a detailed description of the symptoms and the lesions I must refer you to our standard treatises.

The physical signs of capillary bronchitis are as generalized as the lesion; mucous and sibilant râles, of every calibre, are heard together, so to speak. Mucous râles unquestionably predominate, but they are mingled with sibilant, and this in proportion to the plasticity of the products of secretion, which may extend so far as to block totally the calibre of the bronchioles. These râles are in great proportion fine and bursting, and where pulmonary atelectasis occurs these sounds are veiled and may disappear altogether. In the neighborhood the respiratory murmur is in turn altered.

The functional troubles, dyspnea, cough, expectoration, exist in proportion to the extent of the affection. Asphyctic phenomena soon appear; in the infant especially the dyspnea is characterized by thoracic retraction. Cyanosis increases rapidly and death soon ensues.

3. *Chronic inflammatory bronchitis.*—The inflammatory form of bronchitis rarely assumes the chronic type, except when it is dependent on some diathesis or on accidental causes repeated or permanent. Emphysema, with which it is almost always complicated, is a sequela, and in turn contributes to recurrence; during the repeated efforts at cough provoked, air enters the alveoli and is imprisoned in the capillary bronchi by the muco-purulent secretion; thus is produced emphysema as well as dilatation of the bronchi.

Finally there is a point which must be insisted upon, and this is the readiness with which in these patients there occur attacks of acute congestion, which, infrequent and transient at the outset, become more and more frequent and prolonged and aggravate the condition of the patient. In the beginning the patient simply coughed and expectorated abundantly enough each morning, and could attend to his business the rest of the day, only stopping when the retro-sternal pressure, the dry and persistent cough and the acute fever marked the onset of a congestive attack. Soon he becomes a permanent invalid in view of the constant presence of these phenomena rather than owing to the acuteness of the fever.

4. *Inflammatory bronchitis in children.*—Although of less frequent occurrence in infancy than the congestive form, the inflammatory is especially met with in extension and in generalization as a capillary bronchitis, and sympathetically with well-marked dyspnea and even asphyxia. It may be dia-

thesic; it seems often to be connected with the appearance of cutaneous affections more or less irritative. Often also it is accidental and connected with catching cold, something to which the infant is peculiarly exposed.

There are two elements which render the prognosis of this affection much more grave in the infant than in the adult. In the first place, in the infant hematosis is normally more active and the causes at the bottom of it are much more quickly dangerous. Further, in infancy, the calibre of the bronchi being much narrower their obstruction takes place more readily from the presence of the products of secretion.

5. *Inflammatory bronchitis in old age.*—Far less frequent in the old than catarrhal bronchitis, the inflammatory form affects at this period of life in particular the chronic type with more or less abundant muco-purulent secretion. Above all are acute attacks of congestion likely to occur, and it readily passes into broncho-pneumonia. Finally it often assumes the type of hypostatic bronchitis.

6. *Inflammatory bronchitis of pulmonary origin.*—Inflammatory bronchitis may follow on catarrhal, and especially congestive bronchitis, but it is often dependent on affections of the pulmonary parenchyma. In pneumonia, even the most localized form, there exists almost always a certain zone of congestion and even of inflammation. Emphysema, which is commonly a result of persistent or repeated bronchitis, constitutes a predisposing cause of the congestive and inflammatory attacks of bronchitis.

In the tubercular, the bronchitis, which, we have already noted, surrounds the granular foci with a zone of congestive and of catarrhal irritation, may also cause the inflammatory form. It then constitutes one of the most important elements in the evolution of the affection; whether seated in the mucous membrane of the bronchi or in the parenchyma, congestion, and more likely still inflammation, cause an accumulation of elements around the morbid focus, the more or less imperfect organization of which tends readily towards granular and cheesy degeneration. And since, of these processes, inflammation is the chief one which produces these neocytes in greatest number, it is also the process which we must fear the most and combat most actively. Further still, the passage of the products of expectoration over the inflamed bronchial mucous

membrane tends to cause the development of ulceration and thus to multiply the sources of intoxication, whether they be purely septic or specifically tubercular. From my position as physician to the so-called incurable aged at Ivry, I have noted this alteration in a number of old bronchitics, and I showed two specimens to the *Société Médicale des Hôpitaux.* There occurs a shedding of the epithelium and a superficial erosion of the mucous membrane, and this is met with in particular at the level of the bifurcation of the large bronchi and the trachea, the point where the expectoration most likely lodges and is checked for some time in subjects weak enough not to be able to eject it at once. Now these ulcerations are likely to form in the tubercular, since at certain periods of their disease they are as debilitated as possible. Whence the importance of fighting in them every form of bronchitis and of assisting the expectoration, in order to forestall the formation of the inflammatory bronchitis, simple or ulcerative.

7. *Inflammatory bronchitis dependent on pyrexia or on diathesis.*—In the fevers, and notably in typhoid fever, bronchitis rarely assumes the purely inflammatory type, although it frequently approaches it. The same does not hold true for measles where bronchitis, at first catarrhal, readily becomes congestive, and often, particularly towards convalescence, more or less inflammatory. This is the form of bronchitis which yields the nummulary expectoration, which is frequently enough met with at the end of measles.

As for the diathesic bronchitis, we have seen that it is especially congestive in the arthritic and catarrhal in the scrofulous, but when it is chronic, in the one or in the other, it commonly assumes the inflammatory type, at least in repeated attacks, if not permanently, and the characteristic symptom is the more or less purulent expectoration.

LECTURE XI.

INFLAMMATORY BRONCHITIS—DIAGNOSIS AND TREATMENT.

INFLAMMATORY bronchitis may be divided into an acute and a chronic form.

When the bronchitis is purely inflammatory, accidental, like the form produced *a frigore,* its activity is marked and all the signs of congestive bronchitis are present, the fever being intense and permanent. The affection may last from a few days to five or six weeks. It ends in cure, or repeated attacks terminate in the chronic form.

Finally, when it becomes deep and capillary it may risk the life of the patient; and even though it do not expose him to this immediate risk, it is always serious by the traces which it leaves behind, such as emphysema and dilatation of the bronchi.

These results contribute to give to new attacks of inflammatory bronchitis the chronic type. But the chronicity depends above all on the nature of the causes of the bronchitis, and also on the diathesic state of the patient in whom it developes. In a word, it is especially the etiological conditions which govern the progress of the disease.

The *diagnosis* of inflammatory bronchitis depends above all on a characteristic sign drawn from the nature of the expectoration.

The purulent expectoration, or strictly the muco-purulent, signalizes the occurrence of inflammatory bronchitis. Add to this the sensation of retro-sternal pressure which exists, and especially the febrile state, which is the signal of a true inflammation, with high temperature, pulse hard and vibrating, and you have the elements usually sufficient for diagnosis.

Congestive bronchitis, true enough, is accompanied by a more marked and more painful retro-sternal sensation. It is accompanied, further, almost always by vibrating and sonorous

râles, and scanty, viscous, transparent expectoration, often *nil;* finally the evolutionary fever is less continuous, the pulse fuller, and the perspiration greater.

Catarrhal bronchitis points to an irritation, local, less acute, less painful; its râles are moist, abundant, its expectoration easy, fluid, more or less clear; finally, its fever, often intermittent, causes less heat, the pulse is full, perspiration often profuse.

The nature of the râles and their generalization will be, with the dyspnea, indices of the transition of the bronchitis to the capillary bronchi, and should put us on our guard against the concomitant risk.

Treatment of inflammatory bronchitis.—The therapeutic indications do not differ much from those on which we have already laid stress in connection with the two forms, the congestive and the catarrhal. The inflammatory irritation is indeed often only a degree more in the pathological scale, and while calling for the same therapeusis as the lesser degrees of irritation, it requires that these indications should be satisfied with more energy in order to obtain a more effective result. Further we must note that it is the indications of acute congestion which prevail in acute inflammatory bronchitis, and those of chronic catarrh which predominate in case of chronic inflammatory bronchitis, and of this you will be readily convinced in the course of the therapeutic *résumé* on which we are about to enter.

In the acute form of inflammatory bronchitis we will use at the outset emollients; emollients applied in particular on the skin and to the respiratory mucous membrane in the shape of fumigations, pulverizations and even simply of emollient drinks. We need not hesitate here to make the patient drink abundantly, because the hyperemic *rôle* is *nil*, and in the congestive period it is absent. Now hot drinks, by furnishing to the bronchi the elements of a secretion which may directly diminish the congestive afflux, and by provoking a revulsive sudorific movement on the part of the skin, these drinks, I repeat, can only act favorably on the disease in its entirety.

After the emollients come the revulsives—the cutaneous and intestinal. In the inflammatory form it is necessary to employ them more rapidly than in the congestive and more intensely as well. Irritating frictions may thus be dismissed

and wet and dry cupping resorted to at once; blisters are especially useful, large, and placed alternately on one and the other side of the thorax, in front and behind, with the precautions I have already noted in order to avoid the accidents which cantharides is likely to evoke.

In case of the intestinal revulsives, the dialytic purgatives, to which I have hitherto called your attention, are not alone to be used, but also the neutral salts and even certain drastics.

I must here add a few words about venesection. This is to-day rarely, too rarely resorted to. Nevertheless it may be of signal service, especially in the inflammatory and the deep forms of bronchitis. Next to wet-cups, leeches may be applied to the base of the thorax, but they do not always act quickly enough. Venesection, on the other hand, is a powerful means, and when not absolutely contra-indicated by the state of the patient, it may be productive of excellent results. Venesection diminishes the beginning venous stasis, lowers the vascular tension, and by thus making in the chest place for the introduction of air, it favors hematosis. I have seen cases where it has been productive of immediate benefit like that which comes to the individual about to be hung when the hangman cuts the rope, and thus brings on asphyxia. If it be true that venesection does not cure it is just to add that it eases much, that it diminishes the risk and gives us time to use other agents; you have seen this year in our service two cases, certainly one where venesection was productive of the above effects.

Finally in these cases it will often be necessary to resort to nauseants and even to emetics, which are so efficacious when it becomes essential to modify profoundly the pulmonary circulation. We should not delay to administer quickly, in quite large dose, the nauseants which combine a decongestive with a powerful alterative action; such are the antimonials, kermes, white oxide, and even tartar emetic. I will not dwell on the best way of administering these drugs, for I have already dwelt on this; I will recall to you only the fact that we are dealing with a disease in which the nutritive activity of the elements is deeply affected, and, therefore, the treatment must be radical in dosage and in persistence.

The second indication is, we have said, the inflammatory secretion, so to speak. We are no longer dealing, as in case

of the catarrhal form, with a simple hyperemia, but with a plastic production or a multiplication of neocytes. Our aim in the presence of such a process is to alter it into a simple hyperemia; for whatever tends to increase the dryness of the mucous membrane increases its congestion and awakens still further the inflammatory tendency. This is the reason why the agents which favor secretion, and of which I have been speaking to you, are those which are indicated in case of inflammatory bronchitis. We must not check at once the secretion of the mucous membrane, but at the outset modify it and then moderate it. At the outset, hence, we should order soothing drinks and emollient or slightly aromatic fumigations; to these should be added the agents already noted under the head of congestive bronchitis. This result once attained we must not be unmindful of the necessity of evacuating the products of secretion; this we accomplish by means of the expectorants, and possibly by emetics.

In case of capillary bronchitis the marked afflux both of the blood and of the products of inflammatory secretion calls urgently for depletants and for evacuants. The wet cup and venesection are here indicated, and these, together with emetics, in repeated dosage, will clear the bronchial channels.

The cyanosis and the rapid asphyxia of those suffering from capillary bronchitis has suggested the use of inhalations of oxygen. But when we remember that it is not air which is lacking, but the room for it in the thorax, it is readily apparent that what is above all necessary is to let the air channels free. You have seen in my service a number of patients to whom, in analogous conditions, I ordered inhalations of oxygen, at the request of some of your number, and the result has been practically *nil*, even as I had predicted it would be.

Often again, you will have to direct your attention to the general febrile state of these patients and to act directly against this by antipyretics. Finally the essential cause of the bronchitis may be the object to which your therapeutic efforts are to be directed.

In chronic inflammatory bronchitis alterative medication is called for, such as I outlined under catarrhal bronchitis; but with this difference, on which I insist, that we must act with greater force and persistence owing to the greater depth of the nutritive disturbance which we are facing and owing to

the obstinacy with which it persists when once it has obtained foothold in the mucous membrane.

Typical Methods of Treatment Applicable to Inflammatory Bronchitis.

Acute inflammatory bronchitis in an adult.—Order abundant emollient drinks, hot. Apply to the thorax fifteen to twenty dry cups, and four wet cups to the base. Paint with iodine between the shoulders, or apply a large blister for six hours, and then poultice. Give a purgative of sulphate of soda (30 grammes) in an infusion of 10 grammes senna; then keep the bowels open by pills containing 3 centigrammes of podophyllin powder and 1 centigramme of extract of belladonna.

Or, better still:

℞	Rhei. pulv . . .	5 centigr
	Aloes pulv . . .	5 "
	Extr. Belladon . . .	1 "
℥	ft. pil. No. 1.	

Order a light diet: No solids, milk, soups.

Acute inflammatory capillary bronchitis in an infant.—Administer as an emetic:

℞	Pulv. Ipecac. . .	50 centigr.
	Syrup Ipecac . .	50 grammes

S. One desertspoonful every five minutes till vomiting is induced.

Rub the back with cotton moistened with seven to eight drops of croton oil.

Apply to the thorax a large poultice, or if need be a blister for four to five hours, to be followed by a poultice.

Administer every hour fractional doses of white oxide of antimony.

In case of constipation give an enema composed of:

℞	Infus. Senæ (5 grammes of seeds)	100 grammes
	Glycerinæ	10 grammes
℥	S To be used as an enema.	

Give milk diet, alternating with weak milk punch or Malaga wine.

Inflammatory capillary bronchitis in a stout woman at the time of the menopause.—Apply to the base of the thorax twenty-five dry cups. Bleed to the extent of 250 grammes and repeat two to three days after if suffocative attacks recur. Apply mustard plasters to the lower limbs. Small doses of ipecac every two hours. If need be, a blister to the sub-clavicular region and five hours after a poultice. Rigid diet: hot stimulating drinks; milk, soup and wine.

Inflammatory capillary bronchitis in an old man.—Emollient drinks; fractional doses of kermes, repeated; rub the back with:

℞ Liniment camph. ammoniat.
Essence terebinth: . . āā 50 grammes

Under the clavicles apply a large blister (20 centimetres by 8 to 10.)

Diet of milk, punch, soup, Burgundy or Malaga wine.

Chronic inflammatory bronchitis.—Small doses of Dover's powder daily; once or twice weekly order:

℞ Carbonate of magnesia . . 4 grammes
Tartaric acid . . . . 2 "

Fumigations of chlorhydrate of ammonia.

Four to five pastilles of kermes daily, or one to two of calomel each morning.

Revulsive frictions to back and to thorax. Mild and tonic diet.

LECTURE XI.

INFLAMMATORY BRONCHITIS—DIAGNOSIS AND TREATMENT.

INFLAMMATORY bronchitis may be divided into an acute and a chronic form.

When the bronchitis is purely inflammatory, accidental, like the form produced *a frigore*, its activity is marked and all the signs of congestive bronchitis are present, the fever being intense and permanent. The affection may last from a few days to five or six weeks. It ends in cure, or repeated attacks terminate in the chronic form.

Finally, when it becomes deep and capillary it may risk the life of the patient; and even though it do not expose him to this immediate risk, it is always serious by the traces which it leaves behind, such as emphysema and dilatation of the bronchi.

These results contribute to give to new attacks of inflammatory bronchitis the chronic type. But the chronicity depends above all on the nature of the causes of the bronchitis, and also on the diathesic state of the patient in whom it developes. In a word, it is especially the etiological conditions which govern the progress of the disease.

The *diagnosis* of inflammatory bronchitis depends above all on a characteristic sign drawn from the nature of the expectoration.

The purulent expectoration, or strictly the muco-purulent, signalizes the occurrence of inflammatory bronchitis. Add to this the sensation of retro-sternal pressure which exists, and especially the febrile state, which is the signal of a true inflammation, with high temperature, pulse hard and vibrating, and you have the elements usually sufficient for diagnosis.

Congestive bronchitis, true enough, is accompanied by a more marked and more painful retro-sternal sensation. It is accompanied, further, almost always by vibrating and sonorous

râles, and scanty, viscous, transparent expectoration, often *nil;* finally the evolutionary fever is less continuous, the pulse fuller, and the perspiration greater.

Catarrhal bronchitis points to an irritation, local, less acute, less painful; its râles are moist, abundant, its expectoration easy, fluid, more or less clear; finally, its fever, often intermittent, causes less heat, the pulse is full, perspiration often profuse.

The nature of the râles and their generalization will be, with the dyspnea, indices of the transition of the bronchitis to the capillary bronchi, and should put us on our guard against the concomitant risk.

Treatment of inflammatory bronchitis.—The therapeutic indications do not differ much from those on which we have already laid stress in connection with the two forms, the congestive and the catarrhal. The inflammatory irritation is indeed often only a degree more in the pathological scale, and while calling for the same therapeusis as the lesser degrees of irritation, it requires that these indications should be satisfied with more energy in order to obtain a more effective result. Further we must note that it is the indications of acute congestion which prevail in acute inflammatory bronchitis, and those of chronic catarrh which predominate in case of chronic inflammatory bronchitis, and of this you will be readily convinced in the course of the therapeutic *résumé* on which we are about to enter.

In the acute form of inflammatory bronchitis we will use at the outset emollients; emollients applied in particular on the skin and to the respiratory mucous membrane in the shape of fumigations, pulverizations and even simply of emollient drinks. We need not hesitate here to make the patient drink abundantly, because the hyperemic *rôle* is *nil*, and in the congestive period it is absent. Now hot drinks, by furnishing to the bronchi the elements of a secretion which may directly diminish the congestive afflux, and by provoking a revulsive sudorific movement on the part of the skin, these drinks, I repeat, can only act favorably on the disease in its entirety.

After the emollients come the revulsives—the cutaneous and intestinal. In the inflammatory form it is necessary to employ them more rapidly than in the congestive and more intensely as well. Irritating frictions may thus be dismissed

and wet and dry cupping resorted to at once; blisters are especially useful, large, and placed alternately on one and the other side of the thorax, in front and behind, with the precautions I have already noted in order to avoid the accidents which cantharides is likely to evoke.

In case of the intestinal revulsives, the dialytic purgatives, to which I have hitherto called your attention, are not alone to be used, but also the neutral salts and even certain drastics.

I must here add a few words about venesection. This is to-day rarely, too rarely resorted to. Nevertheless it may be of signal service, especially in the inflammatory and the deep forms of bronchitis. Next to wet-cups, leeches may be applied to the base of the thorax, but they do not always act quickly enough. Venesection, on the other hand, is a powerful means, and when not absolutely contra-indicated by the state of the patient, it may be productive of excellent results. Venesection diminishes the beginning venous stasis, lowers the vascular tension, and by thus making in the chest place for the introduction of air, it favors hematosis. I have seen cases where it has been productive of immediate benefit like that which comes to the individual about to be hung when the hangman cuts the rope, and thus brings on asphyxia. If it be true that venesection does not cure it is just to add that it eases much, that it diminishes the risk and gives us time to use other agents; you have seen this year in our service two cases, certainly one where venesection was productive of the above effects.

Finally in these cases it will often be necessary to resort to nauseants and even to emetics, which are so efficacious when it becomes essential to modify profoundly the pulmonary circulation. We should not delay to administer quickly, in quite large dose, the nauseants which combine a decongestive with a powerful alterative action; such are the antimonials, kermes, white oxide, and even tartar emetic. I will not dwell on the best way of administering these drugs, for I have already dwelt on this; I will recall to you only the fact that we are dealing with a disease in which the nutritive activity of the elements is deeply affected, and, therefore, the treatment must be radical in dosage and in persistence.

The second indication is, we have said, the inflammatory secretion, so to speak. We are no longer dealing, as in case

of the catarrhal form, with a simple hyperemia, but with a plastic production or a multiplication of neocytes. Our aim in the presence of such a process is to alter it into a simple hyperemia; for whatever tends to increase the dryness of the mucous membrane increases its congestion and awakens still further the inflammatory tendency. This is the reason why the agents which favor secretion, and of which I have been speaking to you, are those which are indicated in case of inflammatory bronchitis. We must not check at once the secretion of the mucous membrane, but at the outset modify it and then moderate it. At the outset, hence, we should order soothing drinks and emollient or slightly aromatic fumigations; to these should be added the agents already noted under the head of congestive bronchitis. This result once attained we must not be unmindful of the necessity of evacuating the products of secretion; this we accomplish by means of the expectorants, and possibly by emetics.

In case of capillary bronchitis the marked afflux both of the blood and of the products of inflammatory secretion calls urgently for depletants and for evacuants. The wet cup and venesection are here indicated, and these, together with emetics, in repeated dosage, will clear the bronchial channels.

The cyanosis and the rapid asphyxia of those suffering from capillary bronchitis has suggested the use of inhalations of oxygen. But when we remember that it is not air which is lacking, but the room for it in the thorax, it is readily apparent that what is above all necessary is to let the air channels free. You have seen in my service a number of patients to whom, in analogous conditions, I ordered inhalations of oxygen, at the request of some of your number, and the result has been practically *nil*, even as I had predicted it would be.

Often again, you will have to direct your attention to the general febrile state of these patients and to act directly against this by antipyretics. Finally the essential cause of the bronchitis may be the object to which your therapeutic efforts are to be directed.

In chronic inflammatory bronchitis alterative medication is called for, such as I outlined under catarrhal bronchitis; but with this difference, on which I insist, that we must act with greater force and persistence owing to the greater depth of the nutritive disturbance which we are facing and owing to

the obstinacy with which it persists when once it has obtained foothold in the mucous membrane.

Typical Methods of Treatment Applicable to Inflammatory Bronchitis.

Acute inflammatory bronchitis in an adult.—Order abundant emollient drinks, hot. Apply to the thorax fifteen to twenty dry cups, and four wet cups to the base. Paint with iodine between the shoulders, or apply a large blister for six hours, and then poultice. Give a purgative of sulphate of soda (30 grammes) in an infusion of 10 grammes senna; then keep the bowels open by pills containing 3 centigrammes of podophyllin powder and 1 centigramme of extract of belladonna.

Or, better still:

℞	Rhei. pulv . . .	5 centigr
	Aloes pulv . . .	5 "
	Extr. Belladon . . .	1 "
℥	ft. pil. No. 1.	

Order a light diet: No solids, milk, soups.

Acute inflammatory capillary bronchitis in an infant.—Administer as an emetic:

℞	Pulv. Ipecac. . .	50 centigr.
	Syrup Ipecac . .	50 grammes

S. One desertspoonful every five minutes till vomiting is induced.

Rub the back with cotton moistened with seven to eight drops of croton oil.

Apply to the thorax a large poultice, or if need be a blister for four to five hours, to be followed by a poultice.

Administer every hour fractional doses of white oxide of antimony.

In case of constipation give an enema composed of:

℞	Infus. Senæ (5 grammes of seeds)	100 grammes
	Glycerinæ	10 grammes
℥	S To be used as an enema.	

Give milk diet, alternating with weak milk punch or Malaga wine.

Inflammatory capillary bronchitis in a stout woman at the time of the menopause.—Apply to the base of the thorax twenty-five dry cups. Bleed to the extent of 250 grammes and repeat two to three days after if suffocative attacks recur. Apply mustard plasters to the lower limbs. Small doses of ipecac every two hours. If need be, a blister to the sub-clavicular region and five hours after a poultice. Rigid diet: hot stimulating drinks; milk, soup and wine.

Inflammatory capillary bronchitis in an old man.—Emollient drinks; fractional doses of kermes, repeated; rub the back with:

℞ Liniment camph. ammoniat.
Essence terebinth: . . āā 50 grammes

Under the clavicles apply a large blister (20 centimetres by 8 to 10.)

Diet of milk, punch, soup, Burgundy or Malaga wine.

Chronic inflammatory bronchitis.—Small doses of Dover's powder daily; once or twice weekly order:

℞ Carbonate of magnesia . . 4 grammes
Tartaric acid . . . . 2 "

Fumigations of chlorhydrate of ammonia.

Four to five pastilles of kermes daily, or one to two of calomel each morning.

Revulsive frictions to back and to thorax. Mild and tonic diet.

LECTURE XII.

SPASMODIC BRONCHITIS—VARIETIES AND SYMPTOMS.

I DO not propose to sketch for you the history of all the manifestations of spasmodic irritation of the bronchi; but I wish in this lecture to draw your attention to the relations which unite or approach these diverse affections, and to the pathogeny at their basis.

Doubtless more than one of your number is asking the meaning of this expression—spasmodic bronchitis. Bronchitis means irritation of the bronchi, and by the word spasmodic is generally understood purely nervous affections. Nevertheless all irritations are not inflammatory; even as there are congestive and catarrhal forms, so are there spasmodic. Spasmodic bronchitis, then, is an affection seated in the bronchi, and the cause is a peculiar irritation which affects the spasmodic type.

Asthma is the most symptomatic expression of this affection—that is to say, an affection which has as an immediate cause a neurotic irritation, and in symptomatology produces only nervous accidents or functional disturbances of the air passages.

A pathological variety is nearly allied to it; this is whooping-cough, as Trousseau has pointed out; this affection, probably parasitic, epidemic and contagious, has still a purely neurotic type, at least in its essentials.

The manifestations of asthma may, besides, have their outcome from any point whatsoever of the air passages. Thus, in going from above downwards, we have an *asthma of the nose,* or hay fever, an asthma which also follows on any alteration of the nasal fossæ, from a polyp to the simplest form of coryza. Next comes *laryngeal asthma,* which tallies with laryngitis stridulosa, and which may accompany a lesion of the thyroid body or of the larynx; and finally *bronchial asthma,* which is the usual type.

We have still further the secondary manifestations of

asthma, resulting from a mediastinal tumor and especially from the bronchic ganglia—such as tuberculosis of these ganglia—and from direct and indirect lesions of the pneumo-gastric.

Finally, to complete this tableaux of the manifestations of asthma, we note *cardiac asthma,* due to affections of the heart and to gastric disturbances, notably to dilatation of the stomach.

We could still further multiply these varieties by following the descriptions given by many writers, notably the great nosologist Sauvages; but I have mentioned the most important. Finally, we may approximate to these asthmatic manifestations that peculiar type of respiration which has been described by Cheyne Stokes, and which is characterized by a period of apnea followed by a period of respiration gradually increasing and then decreasing, and thus for the moment simulating the attacks of asthma.

All the varieties of respiration of these morbid states are comparable if not identical with asthma, and the mechanism at the bottom of them is at least analogous to that which produces a neurosis, properly so-called. Therefore we will study it at the outset in its most common and regular form.

Asthma is essentially a neurosis. Indeed, it is an apyretic paroxysmal affection, as well as regards the attacks which are, so to speak, the first element as well as the crises which constitute these attacks. The attacks of asthma are provoked by a sensation, often peculiar for each asthmatic, followed by a period of calm.

The initial sensation may begin from any one of the sensitive organs. The attacks are rarely consecutive to a cutaneous impression; nevertheless, in certain patients the action of cold on the skin suffices to induce an attack. Usually the attack of asthma results from a sensation on the mucous membrane of the respiratory passages, whether of the nose, the larynx, or the bronchi. Thus, an atmosphere loaded with certain dusts or impregnated with certain odors, may excite an attack of asthma. In certain cases a descent to a dense atmosphere suffices to provoke an epigastric and dyspneic feeling of depression, followed by an attack of asthma.

In all these instances the attacks begin from an irritant sensation acting on the mucous membrane of the respiratory

tract. Even as in whooping-cough an irritant even simple pressure exercised over the larynx may provoke an attack, so in asthma, an irritant acting at any part of the respiratory mucous membrane may excite an attack of asthma.

There are instances where, indirectly, the provoker of the attacks reveals all the better the neurotic nature of the affection; this is when the attack is induced by a moral impression, or indirectly through cardiac or gastric disturbances.

Symptoms of an attack of asthma.—The attack of asthma may be divided into a number of distinct phases: 1. A period of dyspnea; 2. One of dry catarrh, or of congestion; 3. One of moist catarrh; 4. A consecutive period, or of emphysema.

The patient first experiences a sensation of respiratory anguish, soon followed by deep and repeated respirations during which a considerable inspiratory force is exerted. Little by little the attack developes and the dyspnea increases to such a degree that the patient brings into play all the respiratory powers at his command. The diaphragm, the muscles of the neck, those of the thorax, all take part in the efforts, and determine an active, deep inspiration, accompanied by a species of whistling of the air which penetrates painfully into the respiratory passages, and which is followed at once by a passive expiration, prolonged, and accompanied by râles of varying nature. The contrast is marked between this inspiration, so laborious and brusque, and this prolonged expiration relatively imperfect, and causing only an imperfect emptying of the air which the inspiration has caused to accumulate.

During these efforts the dyspnea is extreme and increases for awhile up to orthopnea. The patient stands erect or in a half sitting posture, the arms extended and the hands fixed tightly to the end of his bed, for example, in order to obtain a fixed point of resistance to the muscles which, attached to the shoulders and to the thorax, effect the distension of the lungs. This dyspnea is accompanied by inspiratory and expiratory whistling, heard at a distance, and by râles on auscultation, râles which are vibrating and sonorous at the outset and later are transformed little by little into moist and sub-crepitant of varying calibre.

The cough, amounting to but little at the outset, increases as the catarrhal stage replaces that of dry congestion; at first the cough is accompanied by a scanty viscous expectora-

tion, that of bronchial congestion, and then the expectoration becomes catarrhal, abundant, white, and still remains painful.

The physical signs are not especially marked: On percussion there is universal tympanites, marked in particular under the clavicles. On auscultation we hear a roughened, choked sound on inspiration, which seems to emanate entirely from the larynx, and at the same time sibilant râles, few in number, are heard, shortened like the inspiration itself; in the succeeding period are heard the sub-crepitant râles, which become moister and larger as the attack nears its end.

Thus it is that the attack of asthma progresses and terminates, the catarrhal period being a trifle prolonged and then disappearing, leaving behind only a slight emphysema which each attack tends to aggravate to a degree. In short, the attack generally beginning in the middle of the night, the patient finds himself at dawn usually in the grasp of a complete catarrh; often, indeed, the crisis lasts only one or two hours; usually it occupies most of the night and too often more still.

Pathogeny of asthma.—It is not my intention to give you the history of the theories which have been invoked to explain and to interpret the nature of asthma—humoral theories, congestive theories, nervous theories, from Galen to Beau. These have always held a marked place in the history of asthma; sufficient to-day if we cite the names of Van Helmont, Trousseau, Lefevre, Todd.

It is to-day not at all in dispute as to whether asthma is of nervous origin or not. But although the affection is a nervous one, it is still to be settled whether it is spasmodic or paralytic. Is it a spasm of the respiratory powers alone which produces the crisis the chief characteristics of which I have recalled to you? Is it a deep paralysis of some other element presiding over this function? These two questions remain to be answered, and each has strong points in its favor.

It is self-evident, indeed, that in the asthmatic crisis there is an inspiratory spasm. If you examine carefully the asthmatic, if you read again the description which I have just given you, you will also be convinced that there exists as well an expiratory paralysis. All the inspiratory agents are brought into action at the time of the inspiratory effort, and coincident with expiration, the expirators remain inert, and the thorax remains almost filled with the air which inspiration

has accumulated, so that renewed inspiratory efforts can cause the entrance of only a small quantity of air, and at the end of a certain space of time cyanosis occurs and signs of beginning asphyxia show themselves in the unhappy being whose thorax is filled with air and yet he cannot renew it nor can he expel it, the alternative motion which causes the circulation of air in the thorax being lacking.

Whence, then, why the dual condition of inspiratory spasm and respiratory paralysis play a strong *rôle* in the phenomena of an attack of asthma. But whence comes the double pathological complexus so contradictory in appearance?

Whence, at the outset, comes this curious inspiratory effort during the attack of asthma, marked by the inspiratory whistling and also by the struggle of the peri-thoracic muscles? The larynx offers no proof of being affected. There is no obstacle to the entrance of air, and if the laryngeal noise suggests difficulty in the passage of the column of air through the larynx, this is not due to stenosis of the larynx, for this is not present, but is due to the fact that the call for air is too great for the orifice which it must traverse. Soon a new source of struggle is added to the first: the air when once in the thorax remains there, owing to the inertia of the expiratory muscles; the necessity of respiration causes the patient to endeavor to make fresh air enter into the thorax, and, since the room is occupied by the air which has not been expelled, the patient wastes his strength vainly in the endeavor to force a minimum quantity of air into bronchi which are already filled. The little air thus introduced does not replace what preceded it, but accumulates in addition, and thus increases the fullness of the thorax and causes emphysema of the lung without aiding hematosis to a great degree.

There, you say, is an explanation which takes well into account all the phenomena; but where find the key to this group of symptoms? What, in a word, is the pathogeny? Whence the partial paralysis and the partial spasm, which are opponents, as it were, in the attack of asthma?

You recall, doubtless, that the pulmonary plexus, like the cardiac, is composed of nervous elements of duplex origin; that is to say, there are present filaments from the great sympathetic and filaments from the tenth pair, otherwise called the pneumo-gastric. Now it is established with cer-

tainty experimentally that, in the cardiac plexus, the pneumogastric compared with the great sympathetic plays the *rôle* of a moderator, and that in certain instances of great excitation of the tenth pair the heart is so well checked that it stops and ceases contracting. Now, this antagonism, or, if you wish, this alternative action of the two systems of nerves in the central circulation is also met with in case of respiration. The two systems of nerves are met with in the pulmonary plexus even as in the cardiac plexus; and Claude Bernard has proved clearly that there as well is found the paralyzing or checking action of the vagus on the sympathetic filaments. In the presence then of a condition which modifies the equilibrium of this duplex action, and the physiological equilibrium being broken, the attack of asthma may occur.

In the normal state the sensation of the need of respiration, when it reaches the respiratory centres, is transformed by them into a motor impression, is transmitted to the respiratory agents by the different nerves of the muscles, the diaphragm, etc., and the lung distended by these powers inspires the external air. In regard to expiration, the thoracic muscular forces are less efficacious; the lung itself acts through its own muscles. Its fibres play the principal *rôle* in the expiratory act, and empty the alveoli and the bronchi of the air which has entered. In short, the active thoracic agents and those of the lung play their part by assisting one another in the two phases of the respiratory act. That is to say, in inspiration the muscles of the thorax are all-powerful, and the muscles of the lung scarcely act at all; in expiration the muscles of the thorax play scarcely any *rôle*, expiration occurring often passively by the return to rest of the inspirators, but the muscles of the lung are in full activity.

This activity of the essential muscles of the lung is under the dependence of filaments from the great sympathetic. It is these filaments which start these muscles at each expiration, even as they start into action the cardiac muscle at each systole. The pneumo-gastrics also act on the filaments of the great sympathetic going to the lung, even as they do on the heart, by suspending its action, so that the abnormal excitation of the pneumo-gastric will stop the lung in expiration, even as it does the heart in systole; under the influence of this arrest the heart remains in diastole, and the lung stays in a condition of inspiratory dilatation.

After these remarks, we may resume the pathology of asthma in the following words:

1. The starting point is generally in a peripheral sensation, and usually in a sensation arising from the mucous membrane of the respiratory tract, that of the nose, of the larynx or the bronchi. Often, indeed, as we have stated, the sensation seems to start from the digestive tract.

2. Whatever the case, the impression received by the organs is transmitted by the sensitive fibres of the pneumo-gastric to the respiratory centre in the medulla.

3. This centre starts the impression along the motor branches of the same pneumo-gastric nerve.

4. Finally, this impression reaches the sympathetic, checks its action, and the attack of asthma occurs.

The initial sensation, whether felt or not, shows itself by a dyspnea which is the first act of the attack. The suspension of respiration explains the rapid increase and the extreme nature of this dyspnea, as also the absence of all expectoration notwithstanding the attempts made by the sufferer. Under the influence of the stasis which ensues, the mucous membrane, irritated by the air which is altered and charged with carbonic acid, begins to secrete with greater or less abundance, at the same time that this secretion is about to be provoked by the paralysis of the sympathetic.

Numerous elements, we see, act as causes in the pathogeny, but the principal are: pulmonary or peripheral impressionability, the medullar reflex, and the indirect action of the motor filaments of the pneumo-gastric on the fibres of the sympathetic. These nervous elements, by reminding us of the nervous character of the disease, enable us to understand how in the attack of asthma inspiration is so difficult and expiration so limited. All the phenomena of the attack are readily explained under such a pathogeny, as well the respiratory origin of the attack as the cardiac and the gastric origins, in dyspeptics for instance.

I have desired to thus sketch for you this pathogeny, which has never, as far as I am aware, been so exposed, although all the facts on which it rests are known and established. I have dwelt on it because it may throw light for you on numerous affections which are allied more or less closely to spasmodic bronchitis.

LECTURE XIII.

SPASMODIC BRONCHITIS—VARIETIES, DIAGNOSIS, THERAPEUSIS.

We are familiar now with the pure type of spasmodic bronchitis—asthma. We must now pass in review the different varieties of this affection, and of those akin to it, in order to group them even as I have enumerated them.

Let us consider, at the outset, the asthma which we may call *asthma of the nose,* known also as *hay fever,* because some writers, noting its appearance year by year at harvest time, have thought that the dust of ripened plants present at this time in the atmosphere was the provoking agent of the symptoms.

Many kinds of dust would seem to be able to play the same *rôle,* since writers cite the most varied. During my term of service as interne I knew a member of a religious community who suffered from asthma, and in order to provoke an attack it sufficed to approach her with a cachet of ipecac in one's hand. Most curious facts are related by writers, and it would seem that the imaginative faculty of the patients was not always an element foreign to the attacks.

This species of spasmodic and catarrhal affection often assumes the proportions of a very painful disease; and when it affects infants, and this occurs frequently enough, it tires them out to a great degree. It is nevertheless an affection absolutely afebrile and frankly paroxysmal; consequently it has all the characteristics of an irritative neurosis. It lasts about five to six weeks and ends by gradual diminution in the intensity of the attacks.

Thymic or *laryngeal* asthma is more chronic and more persistent; it is also eminently paroxysmal and apyretic. It is connected with the irritation of the larynx by the presence of one or another variety of tumor, and has at times received the name of suffocative goitre. Indeed, the symptomatic

form of these attacks depends certainly more on the manner of innervation of the larynx than on the irritant cause, for the spasmodic phenomena are seen in many forms of laryngitis, particularly in *laryngitis stridulosa* and in croup. In a report presented to the anatomical society on foreign bodies in the larynx I endeavored to prove the paroxysmal nature of the phenomena in all such cases.

Anything which can interfere with, irritate or compress the filaments of the pneumo-gastric nerve give rise to bronchial attacks similar to those of whooping cough, and which are allied to spasmodic bronchitis on account of their chronicity, apyretic nature and paroxysmal progress. Such are all the mediastinal tumors, including aneurism of the aorta. The ganglionic tumors are the most frequent, in particular the hypertrophies and those of tubercular nature.

Whooping-cough is an affection which has absolutely the same characteristics and is marked by spasmodic and catarrhal phenomena. Typical whooping-cough differs unquestionably much from asthma, and yet between the two there are many points of analogy; the attack is composed of a series of spasmodic expirations followed by a whistling inspiration, characteristic of spasm of the larynx. The attack ends also by a catarrhal secretion in which both the bronchial and the digestive mucous memoranes appear to concomitantly take part.

All the lesions of the *pneumo-gastric nerves* may also provoke attacks of asthma, and this too no matter at what point the nerve is affected. We may even meet with asthma dependent on a central or bulbar lesion; in which there is lacking the passage of the initial aura or of the sensation which is going to awaken the central reflex; the impression may even arise directly from this centre without any apparent provocation coming from the periphery or the viscera.

Finally, attacks of asthma are met with, alternating with attacks of the *major neuroses.* Van Helmont long ago noted the relations between certain forms of asthma and epilepsy; Trousseau saw the same and has referred to it in his lectures. I myself saw a patient in whom the asthmatic attacks were manifestly alternative with varied attacks, generally epileptiform and probably hystero-epileptic in character.

I have already pointed out to you how severe cardiac dis-

turbances give rise to an impression which goes to the source of the pneumo-gastrics and awakens what might be termed asthmatic manifestations. Whence *cardiac asthma,* often confounded with symptoms peculiar to cardiac disease, because it brings into play the same apparatus, although of different pathogenesis. The point of occurrence of the reflex is in these cases in the heart, instead of in the bronchial mucous membrane; but aside from this peculiarity in origin, the reflex still reaches the medulla, is passed along the motor fibres of the pneumo-gastric, and thus the sympathetic is inhibited.

It will doubtless be asked why the inhibition is produced on the pulmonary instead of on the cardiac sympathetic? This depends on conditions beyond our ken, like the majority of those which determine, in general, the localization of the disease. I know a patient, a sufferer from a mild form of chronic aortitis, in whom there occurs at times cardiac angina and then spasmodic dyspnea, and this should here be called *aortic asthma.*

Let me remark just here that this word *cardiac angina,* I would like to see substituted for the expression *thoracic angina* (*angina pectoris*), which is as bad a word as it is possible to select, and would belong rather preferably to the attacks of grave pulmonary asthma than to the cardiac symptoms to which it is applied.

Still another word in regard to gastric asthma. This is the kind which troubles dyspeptics. Here it is the stomach from which the reflex starts. Since the dyspeptic keeps his bolus for a long time in his stomach and makes chyme but imperfectly, there results a true fermentation; there ensues in consequence a bad effect on the nerves of the gastric mucous membrane. This impression is transmitted to the medulla, and reflected even as happens in the instances of which we have already spoken. Attacks of asthma due to indigestion have been described in the infant (Seé).

These are curious examples of what I purposely term *errors of the pneumogastric.* That is to say, that between the three chief branches of this nerve—the cardiac branch, the pulmonary branch, the gastric branch—there occurs frequently enough an error in transmission which seems to attribute to one of these viscera what belongs to another.

Some among you may remember having seen in my serv-

ice a woman who, affected with emphysema and cardiac disease, complained often in the morning of having had an attack of smothering during the night, and this was not in order to frighten us. On questioning the patient in regard to this symptom the following answer was obtained: she was hungry, she said, and she could only quiet the sensation of smothering by eating a piece of bread, which she carefully kept each evening for that purpose.

What was the truth with this patient? The impression arising from the cardio-pulmonary filaments of the pneumogastric were transmitted by them to the centre, and this centre perceived the sensation as if it had come by the gastric filaments. Now the inverse error is more frequently seen; this is when an impression arising in the stomach is attributed to a disturbance of the heart or of the lungs. Whatever the case these exchanges of impression and of reaction are frequent among the component branches of the tenth pair, and this suffices to explain to us why in many asthma is associated with gastric disturbance.

Further, these localizations may be explained on the theory of special local susceptibility, innate or acquired; *pars minoris resistentiæ.* In case of the nervous system, in which disturbance of function is so frequent, nothing is more simple. The road once travelled by the nervous influence, it tends to follow it again, and thus is the neurosis established.

I have as yet said nothing to you in regard to another form of asthma, to which the name of *renal asthma* has been erroneously applied; you know how likely albuminuric patients are to attacks of more or less intense dyspnea; but the pathogenesis of these attacks scarcely allows us to confound them with spasmodic bronchitis or still less with asthma. Renal dyspnea is produced in those forms of albuminuria which are thought to be dependent on an alteration of the blood, and which, for want of a better name, have been called uremic. We are dealing with a true intoxication; whether this acts directly on the pulmonary mucous membrane through the elimination of urea by the bronchi under the form of carbonate of ammonia; or whether it acts through the alteration in the blood which makes it useless for hematosis; or whether, finally, it acts by excitation of the medulla which is irrigated

by the diseased blood, it is always the alteration of the blood which dominates the pathological scene.

Finally, all writers are about in accord in recognizing a diathesic origin in case of a great many instances of asthma and of spasmodic bronchitis, although they differ both in regard to the nature of the diatheses and in regard to the mechanism more or less immediate by which they determine these bronchial troubles.

The most marked among these pathogenetic influences is without doubt the gouty diathesis; but there is another pathogenetic relation, diathesic or not, which these bronchial affections often assume; it is a relation with chronic or subacute cutaneous affections which has resulted in these diseases obtaining the name of herpetic bronchitis. Whatever the interpretation given to these facts—whether we see a sympathetic exanthema or a substitutive metastasis, the fact remains true and do not forget it.

Diagnosis of spasmodic bronchitis.—Three capital factors enable us to make with certainty the diagnosis of spasmodic bronchitis; it is apyretic, it is essentially paroxysmal in its progress, it is provoked by certain conditions out of proportion to the acuteness and the persistence of the symptoms.

Congestive and catarrhal bronchitis may certainly be apyretic, but then they are light and not at all acute; they may also present remissions and acute recurrences, at times even a true remittence, but the attacks are febrile and are not at the same time dyspneic and paroxysmal. Finally the progress of these affections will always help in differentiation. The phenomena of the attacks give to the neuroses a physiognomy which can scarcely be mistaken.

Congestive bronchitis can, of course, be accompanied, especially when at its acme, by great dyspnea with suffocation; but such an occurrence is transient and it does not recur as in case of the spasmodic affection. Finally, congestive bronchitis is a dry bronchitis as long as the congestion is stationary.

In catarrhal bronchitis, on the contrary, the expectoration is profuse from the start, and it does not pass through the successive stages that characterize asthma. Finally, catarrhal bronchitis, even though remittent, is not in its recurrences marked by spasm; it is further febrile and of limited duration.

We might more easily confound an attack of capillary bronchitis or of suffocative catarrh with an attack of asthma; but the general condition, apyretic in the second instance, and especially the progress of the disease, will not allow us to remain for long in doubt.

A more obscure problem is to find out whether the neurosis is simple; or if not uncomplicated by some organic affection, with what lesion it is combined and what *rôle* these two elements separately play. This problem is especially applicable to pulmonary emphysema. It is above all important to find out in what degree this exists in the dyspneic generally, and in the asthmatic in particular; for it is always met with to a degree, and it appears that if asthma may determine emphysema so too does emphysema often contribute to the development of asthma.

You are familiar with the physical signs of emphysema, the projection of the thoracic walls, the special sonorousness evoked on percussion, and the respiratory noises which are heard, the *piny bruits*, as I term them, since they recall the noise made by the wind as it blows through a growth of pine trees, bruits which vary even unto the diminution or the entire suppression of the vesicular murmur. I need not dwell on this matter further.

Treatment of Spasmodic Bronchitis.—Here, as elsewhere, it is necessary to base the therapeutic indications on the nature, the form, the seat of the affection in the bronchi, which are the immediate seat, the nervous system being the mediate; we are dealing with a neurosis, and usually with a diathesic neurosis. What I have said of asthma is about applicable to all the varieties of spasmodic bronchitis.

The seat is in the bronchi and more particularly in the broncho-pulmonary nervous system. Usually, indeed, the attacks of spasmodic bronchitis begin by an irritation acting on the respiratory mucous membrane; there ensues a modification of the circulation and especially of the bronchial secretion, and in the end the nutrition of these channels is more or less deeply altered and there results emphysema. We must, however, remember that the exciting cause of the spasmodic attack may start elsewhere than in the bronchi, the skin, the nose, the larynx, etc., for example, so as to attack it at its source.

The most efficient indication is, as you know, derived from the form of the affection. Here the form is spasmodic. It is evident, therefore, that the first therapeutic indication is the anti-spasmodic.

In these instances we give at the outset the moderating or calming anti-spasmodics, such as chloroform, which may be ordered by inhalation, chloral, the cyanides, and the various bromides. Often it is advantageous to use the stimulating anti-spasmodics, such as ether, musk, camphor, castoreum, assafetida, and the aromatics like valerian. These agents are to be given according to the strength, the tolerance of the patient, and the chronicity and tenacity of the affection.

These various agents are applicable in general to the attack, not in the midst of it, of course, when the physician cannot do much more than use those palliatives which are rather moral than physiological in their effect. During the attack, that is to say, when the spasms recur frequently, these are of value; between the attacks the anti-spasmodics tend to modify the symptoms and to lengthen the intervals between their appearance. As for the choice between the calming and the stimulating anti-spasmodics, we must above all consult the general condition of the patient; often also it will be necessary to change from one to another, the nervous system being the most changeable of all the systems of the body and passing readily from irritation to excitation through debility, if I may so express myself.

The calming medication has often been used to advantage during the attacks. Opium has been given, in particular as laudanum, even in large dosage (Romberg), or hypodermatically as morphine, as M. Huchard has advised, with the addition of a little ether. The agents used in particular are stramonium, belladonna, atropia, lobelia, phellandra, nicotine, not to speak of tobacco, and conium. These various agents have been administered during the attacks. The most easy to use and the most rapidly efficacious is by fumigation. Cigarettes, containing stramonium, belladonna, alone or combined, with or without nitre, may be ordered.

The substitutive medication, strictly so-called, has more effect on the disease; it may often, even, be used to advantage during the attack.

I have seen Cruveilhier use the method advocated by

Ducrot, and which consists in the application of dilute ammonia to the pharynx. There results, in consequence, a violent revulsion which generally relieves the patient, but at the cost of an extreme difficulty in breathing, for which it is well to be prepared.

The internal use of ammoniacal salts, the carbonate and the acetate, give some relief. Seé has strongly urged inhalations of pyridine. Certain asthmatics drink ice water, others coffee. Emetics may be given, and ipecac, in small repeated dose, has been quite effective in the hands of Trousseau. I say nothing about quebracho and euphorbia pilulifera, drugs more hypothetical than certain in their effects, but they may be tested.

I will not refer to venesection, which could only be of use when a congestive element was superadded to the spasmodic phenomena, whether the congestion affects the lung or other viscera. Hot foot baths, or, better still, perhaps, hand baths, are palliative agents not to be despised. Finally, if at hand, compressed air might be of great utility.

The alterative indication comes next, and such agents are useful, especially between the attacks, in order to modify the nutrition of the elements which constitute the reflex chain of spasmodic bronchitis, in order to change their manner of being and of action.

In the first place we have the iodide of potass in large dose. Used formerly by Aubreé, and highly praised by Jaccoud and Seé, the iodide of potass does not act alone as a modifier of the general nutrition; owing to its eliminative action it is voided by the mucous membrane of the upper air passages and there determines a secretory modification slightly catarrhal, which alters the bronchial mucous membrane, and renders it less susceptible to external agents, and also less likely to remain in that state of relative dryness which is characteristic of the first stage of asthmatic attacks.

The antimonials act in about the same way, either as the white oxide, as kermes, and especially as the arseniate of antimony. The arseniates, indeed, belong by rights after the iodide of potass. I have already spoken of the utility of arsenical fumigations; arsenic internally (Hardy, Delpech), Fowler's solution (Niemeyer), the arseniate of soda (Trousseau), are powerful alteratives, and efficacious in case of spasmodic bronchitis.

Finally, all the expectorants are useful adjuvants in these affections—useful, in the first place, because by promoting the secretion of the mucous membrane they modify its sensibility and render it less impressionable; useful, in the second place, because by ridding it of the products of secretion they prevent its becoming dry. The preparations of antimony and of ipecac, already noted, are also of value under this head; squills, gum ammoniac, the use of which I have referred to in previous lectures, also find their indications here. The same holds true of sulphur, which has been advised in small doses (Debreyne), as also the balsams in general, under which I must not forget to rank terpine.

Further still, many of these alterative agents answer our third indication, and the majority have been administered as well as diathesic agents as local alterants; it is from this same standpoint that we must consider the thermal cure of these patients.

I only desire to refer briefly to a few other medicaments which have been more especially used in certain forms of spasmodic bronchitis; thus in whooping-cough, cochenille, the bromhydrate of cicutine. I cannot, for my part, too highly recommend to you this latter drug, from which I have obtained excellent results, as well in hay fever as in whooping-cough, not indeed to the extent of checking the disease, but in that I have always been able to make the interval between the attacks longer and attenuate them to the degree of making the affection very moderate in its symptoms and in its general aspect.

I have not endeavored, gentlemen, to write for you the history of asthma, as you are doubtless aware, but only to give you an exact idea of spasmodic bronchitis in its varied forms, among which asthma certainly holds the first place.

Typical Forms of Treatment Applicable to Spasmodic Bronchitis.

1. *Spasmodic bronchitis in a nervous young man.*—Chloroform liniment over the thorax; order every three to four hours a pill containing 5 centigrammes of extract of datura; order every night, and during the night in case of an attack, small doses of laudanum in cherry-laurel water.

2. *Attacks of asthma; means of utility during and just before the attack.*—*a.* Before the attack order a hot mustard foot bath; burn nitre paper in the room and order a cigarette made after Trousseau's formula: paper dipped in a 10 per cent. solution of arseniate of soda and wrapped around a mixture of datura and of tobacco. Give an injection of chlorhydrate of morphine (.01 to .02 with or without admixture with ether). Inhalations of chloroform may be tried. If the patient can take it administer about 5 grammes of wine of ipecac (Grave's method).

b. When the attack has begun, order a hot hand bath; let the patient inhale ammonia or pure aromatic essence or a mixture of this and of chloroform. Give a little laudanum every ten minutes, or administer an injection of morphine protected by atropine; assist expectoration by small doses of squills and polygala.

3. *Means useful between the attacks of asthma.*—Order before meals twenty grains of iodide of potass, well diluted. Morning and evening order with a pastille of kermes one of the following pills:

℞ Extr. stramon.
Zinci valerianat āā 10 centigr.
℥ et ft. pil. No 1.

Every other day let the patient take a laxative composed of 100 grammes of infusion of senna (10 grammes of the seeds) and 20 grammes of cream of tartar, and on the intermediate day a hot mustard foot bath. The diet should be light and stimulating.

4. *Chronic asthma and emphysema.*—For fifteen days each month, in the morning before breakfast, order about 30 grains of iodide of potass; once a week let the patient purge himself with 20 centigrammes of calomel. Every night, at bedtime, order 2 to 4 grammes of arseniate of soda for one week, and the following week 2 to 4 pills of bromide of camphor (each 20 centigrammes). In case of dyspnea order fumigations of nitre paper in the bed-room, or, better still, Trousseau's cigarettes (nitred paper filled with datura and tobacco, or datura, belladonna, and tobacco). During the two weeks when the iodide of potass is not taken, order 2 to 4 pills of terpine (each 20 centigrammes). Let the diet be light; sup-

press all stimulant; order milk and white meats. Each month let the patient receive about ten séances of compressed air for 1½ hours with ½ an hour of permanent compression. During the summer let him live at Mont Doré.

5. *Hay fever.*—Morning and evening let the patient take three to five granules of bromhydrate of cicutine (each 1 milligramme). Before bed-time let the bed-room be sprayed with:

℞	Tr. eucalyptus	25 grammes
	Acid: Phenic: pur: . . .	50 centigrammes
	Ft. sol.	

At bed-time let him take:

℞	Infus: aurant: flor . . .	80 grammes
	Syr. menth: pip:. . . .	20 "
	Chloral hydrat.	1 gramme
	Sodii bromid	2 grammes
℥	S. At a dose.	

In case the symptoms persist give the iodide of potass.

6. *Laryngeal asthma; spasmodic laryngitis.*—Twice daily let the patient inhale the following decoction:

℞	Aquæ	500 grammes
	Belladon. flor	
	Datura flor	āā 5 grammes
℥		

Let him gargle two to three times daily a strong infusion of leaves of coca and swallow a mouthful every now and then. Morning and evening let him rub his neck in front with chloroform liniment, and later on, if need be, let him substitute the following:

℞	Liniment: ammoniac. . . .	50 grammes
	Ess. eucalypt.	10 "
℥	S. External use.	

Let the diet be light, with no stimulants.

7. *Cardiac asthma; organic disease of the heart; hypertrophy; rheumatic diathesis.*—Each morning between the attacks, iodide of sodium, 20 grains. Each night, before dinner, two teaspoonfuls of:

℞	Infusion *de houblon* . . .	250 grammes
	Syr. aconit	50 "
	Brom. sod	25 "
℥		

At bed-time let the patient take 15 to 20 drops of tincture of digitalis with the same amount of tincture of lobelia. Two to three times daily order 4 to 5 grammes of pyridine by inhalation.

8. *Gastric asthma; gouty dyspepsia.*—Before two of the meals:

℞	Carbonat. magnesiæ	25 centigr.
	Pulv. foll. belladon	25 milligr.
℥	S. At a dose.	

At the end of each meal order a cupful of infusion of coca with syrup of orange skin.

Each morning, on an empty stomach, order:

℞	Syr. menth. pip	50 grammes
	Sod. arseniat.	10 centigr.
	Tr. lobeliæ	5 grammes
	Aquæ pur.	150 grammes
℥	S. A desertspoonful.	

At night, in case of threatening dyspnea:

℞	Extr. stramon.	5 centigr.
	Extr. opii	25 milligr.
	Zinci ox.	5 centigr.
℥	ft. pil. No. 1.	

Morning and night have the neck and chest rubbed vigorously, also over the stomach and liver.

The diet must above all be watched with care in order to banish all food likely to interfere with a weak digestion, and especially those which by fermenting would cause tympanites. At the end of the meal a little stimulant might be allowed. No tea, scarcely any coffee, no liquor, no poultry is to be tolerated.

If need be order malted beer.

8. *Whooping-cough in a child of seven.*—Three times daily give two granules of bromhydrate of cicutine (each 1 milligramme). Increase the dose by one granule daily till 9 milligrammes are being taken. Continue this dose for three to four days, and then suspend for one day, and renew.

Each night vaporize the room with weak phenic acid solution or benzinated water. In case the vomiting persists

notwithstanding these means, give before each meal in a little sugar and water:

℞ Aq. menth. pip. } āā
Aq. chloroformi }
♏ S. One teaspoonful

LECTURE XIV.

THE SO-CALLED INFECTIOUS BRONCHITIS. ETIOLOGY, SYMPTOMATOLOGY, TREATMENT.

I HAD intended concluding these lectures with the last, thinking that I had about completed the circle of the alterations which an irritant may determine on the mucous membrane of the bronchi, when some of you expressed the wish that I should add another lecture on the so-called septic varieties of bronchitis, or the infectious varieties.

In reality, to conclude with infectious bronchitis is the proper way to end the interesting study of the pathology of bronchitis, although unquestionably in doing so we depart a trifle from the primary object we had in view; still there is really nothing contradictory in our action. While recognizing the nosological basis on which rests this variety of bronchitis, it will be easy to show you in the course of the brief remarks I propose making the importance of the physiological element in the special manifestations under which this affection may appear.

From an anatomical standpoint, compared with the common forms which we have studied—that is to say, the congestive, the catarrhal, the inflammatory and the spasmodic—we need only consider three special types of septic or infectious bronchitis: the ulcerating, the gangrenous, the diphtheritic.

I have already spoken of those bronchial ulcerations which occur in case of certain varieties of chronic bronchitis, as the result of the purely mechanical stagnation of the more or less altered products of secretion on the mucous membrane at the level of its sulci. There are still other bronchial ulcerations: that, in the first place, which results from the presence of tubercles in the thickness of the mucous membrane; and that, in the second place, which is met with in tubercular subjects, not so much on account of the development of a local tubercle,

as on account of the nutritive destructive processes to which all cachectics are liable.

In studying the history of the varieties of laryngitis you will meet with these various occurrences and causes of ulceration.

As for gangrene of the bronchi it may appear in different ways which it is essential to differentiate. It may follow in the same causes as those to which we have just referred under the ulcerative type; that is to say, it may result from the arrest of local nutrition and be purely co-incidental, or else it may be caused by a disturbance of nutrition more general and to a change in the blood. It may complicate an organic cardiac or pulmonary disease.

There is a variety of gangrenous bronchitis which especially deserves this name, and which has been described under the name of curable gangrene of the lung, and which in reality is only a superficial gangrene of the mucous membrane perfectly susceptible of cure. The same considerations apply to its etiology and pathogeny.

Finally, we must consider diphtheritic bronchitis, which is associated with the disease of the same name and may also appear under two distinct forms: the one the result of simple extension of the diphtheritic layer from the pharyngo-laryngeal mucous membrane to that of the bronchi; the other, which developes in the bronchi at the outset, without propagation from the neighborhood, following on a general intoxication. The first form, be it well understood, is far less grave than the second; for although it exposes the patient to asphyxiation, even to consecutive intoxication, there is an opportunity for the membrane to become shed before these accidents can occur; while the second form carries with it a general and deep intoxication, and thus increases greatly the necessary risks of the disease.

It might seem appropriate to add to these varieties, without question the most important and characteristic, those which accompany great pyrexia and deep poisoning; but we may say here, once for all, that the different causes of these morbid localizations are likely to produce above all catarrhal bronchitis and the passive congestive form, which we have already studied; or else they approximate to the special forms we have just spoken of, according as they become ulcerative,

gangrenous, or pseudo-membranous. Such are the bronchitis of typhoid fever, of the virulent and infectious fevers—pyemia, for instance, and that which accompanies uremia.

Causes.—The causes of infectious bronchitis may be resumed in a few words, as follows: At the outset cases where the alteration in the nutrition of the bronchi depends on the intensity with which the irritant action affects the mucous membrane; those, again, which depend on the weakness and depressed condition of the general nutrition of the patient; finally, the instances where the alteration depends especially on the toxicity of the morbid cause. I will only add to refresh your memory the nutritive alterations dependent on mechanical compression by tumors of the thorax pressing on the bronchi, on lesions produced from without inwards by the introduction into the air passages of irritant foreign bodies. I have a number of times seen old pleurisies accompanied by gangrenous bronchitis.

Let me relate to you here a case in which I was much interested: A man in full strength and health, but subject to rheumatic attacks, caught cold as the result of a night passed half in an insufficiently heated railway station and half in an open carriage. The resulting pleurisy was complicated by phenomena of gangrenous bronchitis, and of such a degree that not only did the expectoration have a fetid and a gangrenous odor, but the breath exhaled such an insupportable odor that his room and everything in it were infected. The gangrene was not difficult to diagnosticate, and since the signs of pleurisy had not all disappeared, I predicted the possibility of a pulmonary perforation, and this soon happened. The insufficient nutritive state in which this rheumatic was determined this dangerous morbid condition, and he had not sufficient strength left to overcome it.

Symptoms of infectious bronchitis.—Let me resume, in a few words, as I did for the etiology, the symptoms of infectious bronchitis.

The symptoms are, we may say, always secondary, and they arise amidst a different symptomatology according to the primitive disease to which they belong. In any event it is almost always in patients more or less weakened by bad hygiene or by prior disease that we see these varieties of bronchitis develop. All the constitutional diseases are predispos-

ing, but it is above all the soil which is of importance, and the least infectious disease may at any time change into the infectious form in case the patient is in a state of weakened or insufficient nutrition.

The bronchial complication is heralded, generally, by fever, or at least by exacerbation of the fever from which the patient is already suffering, and by an aggravation of the cachectic state, in which he already is.

It is especially the functional disturbances which, in such cases, draw attention to the bronchi. Dyspnea is not always present, and in the state of depression in which the patients are the most marked symptoms are masked and the majority of the subjective signs are wanting. Still, when the bronchi are so obstructed that asphyxia is likely to set in, we see occasionally transient moments of functional wakefulness when there occur attacks of dyspnea even to an intense degree, so much so that asphyxia and poisoning joining their forces, the patient remains in a state of indifference and of ominous anesthesia.

The cough is a constant accompaniment, and in general it pursues the same course as the dyspnea, being acute enough at the outset, at least at intervals, and only ceasing at the end of the process.

The expectoration is here, without question, the most certain element in the clinical picture, and on it in general the diagnosis is based. Usually, and at the outset, it is mucopurulent, more or less catarrhal and inflammatory, according to the primitive variety of the affection which is complicated. When ulceration of the bronchi sets in, the expectoration soon becomes discolored, at intervals, by a slight sanguineous tint, even though the ulcerations are not very deep. Whatever the extent of the process, however, under the microscope are discovered in the expectorated matter, besides the elements which belong to the primitive affection, blood corpuscles and their coloring matter, and also the epithelial cells which are exfoliated from the surface of the mucous membrane.

Almost always, also, when the ulcerative process is extensive, the expectoration has a stale odor which denotes beginning fermentation of the products.

In case of gangrenous bronchitis the expectoration also furnishes the diagnostic sign. The same conditions I have in-

dicated are united, and furthermore the expectoration and the breath of the patient, especially the former, exhale a fetid odor which suggests a macerated anatomical specimen, and which is never forgotten when once it has been smelled; this odor, therefore, is a specific sign of gangrene. The microscope, better still than in the ulcerative form, will show in the sputa the elements of the gangrenous eliminated tissue, with the blood corpuscles which follow on the ulceration consecutive to this elimination.

At times the physical signs will enable us to specify the parts which are particularly involved in the gangrenous process, and this by the aggregation of the moist râles in distinct foci, which allows us not to confound them with the diffuse signs of ordinary bronchitis. Often, indeed, the focus once formed will give rise to a souffle, a souffle of induration resulting from the plastic zone of irritation which forms around it, and further still the cavity souffle, when a void is left by the expulsion of the gangrenous products. These signs, however, are rarely met with because gangrenous bronchitis, at least in its most usual form, has not much tendency to extend into the thickness of the parenchyma of the lung, and rarely leaves other than superficial imprints, so to speak.

As for pseudo-membranous bronchitis, it is, at the outset, marked by considerable dyspnea, and apparently, out of all proportion to the lesion. At the same time occur attacks of suffocative cough, painful, and with a certain inspiratory whistling which is evidently produced at the beginning of the respiratory tract. In fact, auscultation reveals that there exists from the side of the lungs only a marked weakening of the respiratory murmur. It is only when the false membranes tend to become detached and when the bronchi begin to secrete a more or less catarrhal flow, that we hear mucous moist râles generalized and often a crackling noise, as though valvular, due to the lifting up of these diverse products by the passage of the air, a noise which has been compared to the shifting of a curtain by the wind.

The expectoration, in these cases, may contain false membranes, sometimes even true pseudo-membranous casts of the calibre of the bronchi. It is very important to differentiate these products of concreted mucous secretion from fibrinous exudates and the cylinders which result from an intra-bron-

chial hemorrhage, and also from the secretions of aphthæ which are sometimes met with in the bronchi. The microscope will enable us easily to differentiate.

As for the typhoid and the pyemic varieties of bronchitis, these are truly secondary phenomena in relation with the pathological state in the midst of which they occur. In fact no special sign reveals their origin; they are varieties of congestive and of passive catarrhal bronchitis, as I have already stated, which are marked by the characteristics which we have noted, and are distinguished in particular by the general state of the patient in whom they develop, or by the morbid condition in the midst of which they are evolved.

But here again I am unable to give you a complete history of these diseases, and I only desire to indicate to you clearly the practical deductions to be drawn from this review—in a word, the therapeutic indications of infectious bronchitis.

Therapeusis of infectious bronchitis.—I have endeavored to point out to you in the course of the brief *résumé* which I have made, the important place which, in these varied expressions of septicity,the state of general nutrition and the strength of the patient occupies. Nutrition is reduced to a minimum, so that total death or at least rapid degeneration of the anatomical elements occur, and the strength is reduced to such an extreme degree that the patient cannot conceal the loss.

The first indication to be filled, then, is to lift the nutrition up to a higher level and to provoke the return of a more effective and regular activity in the system. At the outset, then, tonics are to be used; and we may state that here tonic medication may be used in all its forms.

The chief agents should be oxygen and the chlorates, which act especially on hemapoiesis; coca, quinine and phosphates, which add resisting power to the elements, and finally alimentary agents capable of easy and rapid digestion, such as milk, liquid foods, bouillon, wine.

To these tonics must be added those which are capable of stimulating the nutritive functional activity. Such are: wine, and in general all the alcholics in fair dose. Coffee, tea, are to be given, and all the anti-spasmodics we have spoken of: ether, valerian, musk, camphor and the aromatic umbelliferæ.

At times preparations of phosphorus are added, with which it is well to be careful, and to recall the fact that if the excitant

dose of phosphorus be passed, the excitability of the elements is rapidly used up, and they become fatty, granular, degenerated.

This tonic-stimulant therapeusis is called for by the condition of the patients; but the local condition of the bronchi calls for a different medication; the nutrition of the mucous membrane must be modified, the nature of its secretion must be altered, and the more or less gangrenous process at work must be checked both as regards the products of this secretion and also as regards the elements of the altered mucous membrane.

This indication is satisfied by the use of substitutives and of antiseptics. All the former class which we enumerated particularly under the head of catarrhal bronchitis (chronic) may be used to advantage here. The balsams in particular, the resinoid preparations (turpentine, eucalyptus, copaiba, cubebs) as also creasote, find here their indication. The sulphurs may also be utilized, at least in chronic, prolonged varieties.

As for antiseptics, the object of their use is evident; it is only necessary to select those which can easily be applied to the respiratory tract. Thymol answers this purpose, and with it, as also better than it, iodoform. Creasote and phenic acid preparations are also of use. All these agents are more valuable if used locally by fumigation or the spray.

Of all these agents those best suited to ulcerating bronchitis are, aside from expectorants and the majority of the drugs resorted to in case of chronic bronchitis, tonics, stimulants, the substitutives; the antiseptics are only of secondary utility here.

In gangrenous bronchitis, on the contrary, the antiseptics hold the first place in the local treatment, as also the balsams, which are not only antiseptic, but which also favor hematosis owing to their oxidizing action. As for the general treatment the simple tonics and stimulants rank above all.

In case of pseudo-membranous bronchitis, a general tonic treatment should be the first indication, the danger lying in the asthenia, which results from the diphtheritic poisoning. Next in order come the substitutives, those in particular which we know are capable of producing on the mucous membrane a catarrhal irritation with moist and abundant secretion, for we have seen that it is often by transformation into this species of irritation that the pseudo-membranous irritation is cured.

Among these therapeutic agents belong all the balsams and the essences in particular; tolu, cubebs, eucalyptol. Under this head have also been given mercurials, in especial repeated doses of calomel, also the chlorate of potass, iodoform, the iodides and the bromides, alcohols, and to complete the effect of these various agents expectorants are administered to cause the expulsion of the products detached from the mucous membrane. Finally, to these agents may be added the sulphurs.

As for the congestive and the passive catarrhal bronchitis of the essential fevers, when they call for medication, the revulsive type may be of utility, as also the means which I have mentioned under the subject of catarrhal and congestive bronchitis; but owing to the passive nature of these complications the dominant therapeutic indication must be a stimulant one, generally stimulant and stimulant in particular of the bronchi. The essences and all the balsamics and alcohols fulfill this indication. The ammoniacals may also be added to a certain degree.

Finally, owing to the general state of the patients, the most stimulating tonics should be ordered.

Typical Forms of Treatment Applicable to Infectious and Septic Bronchitis.

1. *Ulcerative bronchitis following on chronic bronchitis.*—Three times daily order one of the following pills:

℞	Pulv. scillæ.	2 centigr.
	Gum ammoniac	5 "
	Ess. eucalypti	gtt. j.
ℳ	ft. pil. No. 1.	

In case these do not answer, order:

℞	Creasote	gtt. iij.
	Ess. citroni	gtt. ij.
	Aq. puræ	120 grammes
	Syr. aurant. flor	30 "
ℳ	S. A teaspoonful every two to three hours.	

If the patient prefers he may take one to four to five capsules of cod liver oil creasoted.

Sulphurous inhalations may be ordered to advantage.

Before each meal a glass of quinine wine should be taken pure or with the addition of a third or a quarter of glycerin.

The diet should be tonic. Alcohol should be ordered at the end of each meal or a cup of some aromatic infusion.

2. *Gangrenous bronchitis* (*curable gangrene of the lung*). —Three times a day order:

℞	Infus. quininæ	100 grammes
	Syr. aurant. cort	50 "
	Ess. eucalypti	gtt. 5.
	Tr. antiseptic (?)	20 grammes
℥	S. A desertspoonful.	

Let the patient inhale powdered iodoform each night, and have the bed-room sprayed with thymol.

Before each meal order some quinine wine.

Same diet as in preceding case should be given.

3. *Pseudo-membranous bronchitis, in a case of diphtheria with tendency to generalization.*—Every other day the patient should take in the morning fasting, unless he has diarrhœa, the following powder:

℞	Calomel	10 centigr
	Pulv. jalap	2 "
℥	S. At a dose.	

Every hour give a mixture containing chlorate of potass and acetate of ammonia in usual dose.

Three times daily order a eucalyptol perle.

Have the bedroom frequently sprayed with carbolic solution.

Give stimulating drinks, coffee, rum, chartreuse; order a little pepsin, the elixir for instance, and a little quinine, daily. Give extract of beef and peptones by enema, if necessary.

4. *Septic bronchitis with pyemia.*—Order quinine; apply twenty dry cups to the base of the thorax, and renew each day or every other day; give three times daily:

℞	Infus. arnicæ	100 grammes
	Syr. limonis	50 "
	Glycerin	25 "
	Acid: phenic.	1 gramme
	Aq. menth. pip.	20 "
℥	S. A desertspoonful.	

Purge every two to three days with .10 grammes powdered calomel, taken in the morning fasting.

Order each night in very hot punch twenty drops tincture aconite and ten drops laudanum of Rousseau.

Order repeated fumigations with infusion of eucalyptus, pure or with the addition of a few drops of eucalyptol.

Allow coffee, wine, milk, meat, eggs, even as in the preceding cases.

ON

GONORRHŒAL INFECTION

IN WOMEN.

BY

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ON GONORRHŒAL INFECTION IN WOMEN.

CHAPTER I.

GONORRHŒAL INFECTION PRODUCES COMPLICATIONS WHICH ARE SERIOUS IN THEMSELVES AND OF FREQUENT OCCURRENCE.

IMPORTANCE TO THE GYNÆCOLOGIST.

GONORRHŒA as it occurs in the female sex is still in this country strangely neglected by general practitioner and specialist alike. Its symptoms, its differential diagnosis, and the ravages which are its immediate or remote results, are hardly recognized or understood, and the treatment of it, as ordinarily practised, is contemptible. Yet the virus of this disorder gives rise to a group of diseases, a series of pathological conditions, which, by reason of their clinical interest and their social and moral consequences, surpass in importance any other class of affections with which the gynæcologist is called upon to deal. To the reader, cognisant of the eager activity which, now as heretofore, pervades every department of medical research and practice, there must appear to be something extreme or paradoxical in this statement. But the explanation is not far to seek. If gonorrhœa stood alone among the diseases of women that had been neglected for generations by the surgeons, the explanation might be difficult; but it has only shared what was the common lot with abdominal and uterine surgery until the latter was removed from the limbo of neglected subjects by the work of the specialists, so recently that many of the early reformers are still alive and engaged in the active practice of their profession. Even yet the importance of the part played by the gonorrhœal

virus in the production of disease has received comparatively little attention from the gynæcologists in England and America, whereas in some other countries, especially in Germany, in recent years, a large amount of experimental work has been done, and much clinical evidence has been collected, which must soon work a revolution in the opinions and practice of the profession throughout the world in regard to certain phases of gonorrhœal disease. So little even of foreign work has been published in England, that it can be no exaggeration to say that if the whole subject could be put before the practitioners of this country in all its fullness of detail, both theoretical and practical, as it has developed in Germany during the last few years, the knowledge would come as a surprise and a revelation. Writing with this conviction, I need hardly apologise for endeavoring to give an outline of these recent researches and observations, especially as I shall be able to incorporate some observations and illustrative cases of my own, which can hardly fail to be recognised as corroborative evidence in support of conclusions already suggested in the opening sentences, and ultimately to be stated more explicitly and formally.

Erroneous and Inadequate Views Taught.

There are few medical practitioners in this country who, when medical students, were not entirely mistaught with regard to the pathology and treatment of gonorrhœa in women. Their teachers were the pure surgeons, and when these taught that the disease was to be considered only a form milder in its course, and more amenable to treatment, when affecting the female sex, there need to be no surprise if the practitioner has carried the ignorance of his teachers into his daily practice, with disastrous consequences to some of his patients. Whether the teaching of an old pathology is still carried on, I do not know; but there is no evidence of a change in some of the most recent works on surgery. It would be interesting, if space permitted, to collate a series of quotations on gonorrhœa in women from the older and more recent manuals of surgery, as exhibiting the barrenness of surgical observation until the subject was taken in hand by the specialists. It must suffice, however, to quote from the article "Gonorrhœa"

in Holmes's "System of Surgery." This work is selected as one which is looked upon by many practitioners as a complete library of surgical literature, and as I quote from the third edition, published in 1883, it may fairly be assumed that we have here a statement of the orthodox surgical doctrine of the present time, as understood by the average general practitioner; and, having it fresh before us, we shall be better able to see how we stand with regard to the orthodox creed when we have assimilated the newer knowledge. Under the subdivision, "Gonorrhœa in the Female," the author says[1]: "Gonorrhœa is a much less common affection in women than in men, and when it does occur, the disease is ordinarily much less severe, and hence more rarely comes under treatment. . . Inflammation, in consequence of gonorrhœa, has been supposed to extend to the cavity of the uterus and to the Fallopian tubes. West mentions two successive attacks of vaginitis, at an interval of eighteen months, in same patient, which were followed by such severe peritonitis as to call on each occasion for the abstraction of blood. . . . Ovarian inflammation, corresponding to the epididymitis of the male, occasionally occurs. It has been described by Hunter, Dr. Tilt, and others."

There is in this chapter not a single expression to convey to the reader any hint of the serious nature of the complications of gonorrhœa in women, as compared with the corresponding phases of the affection in men. The existence of a "latent" or creeping form of the disease is not so much as mentioned, and the author is equally silent with regard to dysmenorrhœa, pyo-salpinx, sterility, and other well-recognised results of the disease. It will be observed that the date of this essay is eleven years later than the appearance of Noeggerath's work; six years later than when Lawson Tait wrote,[2] "Acute ovaritis from gonorrhœa is a common result of the infection;" and four years after Neisser's first publication on the discovery of the gonococcus. It need not surprise us if the practical outcome of such teaching is a very lame method of dealing with the disease in the male, with unfortunate results for the female. The ordinary treatment of the male sex, with its consequences, is well illustrated by a case at present under my care, which,

[1] "A System of Surgery, by various Authors." Edited by Holmes; p. 391, 3rd Edition, 1883.

[2] "Diseases of Women, 1877.

in its clinical aspect, as affecting the woman, will be given in more detail in the sequel. It is that of an hospital patient, suffering from acute pelvic inflammation, as I shall call it at present, the special internal development of the disease not having been as yet sufficiently differentiated. She has been married only about two months, and she has a turbid uterine discharge, which has been ascertained to contain gonococci in abundance. In order, if possible, to obtain corroborative evidence, without too closely questioning the wife, I sent for the husband. I told him my object in wishing to speak with him was to learn if he had had clap? "Yes, I had," said he, quite frankly. "When?" "I got it in August last year." "When were you cured?" "In November." "When did you get married?" "In the beginning of February." "When did your wife begin to complain?" "Last month"—that is, in March, just about a month after marriage. In answer to further questions, my interlocutor informed me that the doctor who treated him first, considered him quite cured in November. There was some uneasiness in the genitals about Christmas, and he consulted another practitioner, with special reference to the question of marriage at an early date. Although the patient was the subject of phimosis, and his glans could not be seen, he was assured that he was all right, and might get married without misgiving. Very soon after marriage he began to have a discharge, and his wife became ill, complaining of intense hypogastric pain, which necessitated her taking to bed. Still another medical practitioner was called in to attend the wife, and he was also consulted by the husband on account of some symptoms which had reappeared. This doctor recommended, and performed, the operation of circumcision on the husband, and is reported to have assured him that there was no sort of connection between his wife's ailment and his own. The final result in this case, so far, is a gleet in the husband, and an ailment which is probably destined to do irretrievable damage to the sexual organs, and perhaps to the general health, of the wife.

In further illustration of the practice which may result from an inadequate apprehension of the danger to women arising from gonorrhœal infection, I shall quote from a comparatively recent French writer on the venereal diseases. Speaking of the case of a young man about to be married, who has had

the misfortune to contract a gonorrhœa which there is not time to cure before the wedding day, he says: "In these embarrassing circumstances, one sovereign remedy remains, only one—injections of nitrate of silver. . . . Suppose one employs a medium dose of the solution, in an hour and a half or two hours after the injection there appears a discharge, the simple effect of traumatism, accompanied by a little smarting in micturition. This slight inflammation lasts five or six hours. But then, precious result, the canal becomes dry; all morbid secretion is arrested, and this condition persists for eighteen or twenty-four hours—quite sufficient time for the bridegroom to seek his nuptial couch in a healthy, or, at least, noncontagious state. In exact figures, if the newly-wedded pair ought to retire to their room on Friday morning at one o'clock, the preservative injection ought to be made about nine o'clock on Thursday forenoon."[1] This is surgery fit only for satyrs; and yet the practical outcome of much of the prevalent treatment is as inevitably the ruin of the health of innocent women, as must be the result of following the coarse advice of this French surgeon.

Some who have paid but little attention to the diseases of women may consider the terms here employed to indicate the effect of the venereal poison upon the health of the woman unnecessarily strong. But it will be shown later on that the gonorrhœal virus may, and does frequently, cause chronic discharges, metritis and endometritis, salpingitis and pyosalpinx, ovaritis, peritonitis, derangements of menstruation, sterility, and a variety of complications and combinations of these diseased conditions; and, if this can be proved, any language fails to do justice to the facts. It may be also objected that such severe cases must be rare, and therefore the subject becomes comparatively unimportant. The frequency with which the more serious cases occur appears to vary in different countries—that is, as might be expected, according to the habits of the people. The apparent frequency also depends upon the method in which the facts of the history of each gynæcological case are investigated—that is to say, the nature of the cases is overlooked or detected according as the practitioners are ill or well informed on this special theme.

[1] "Le péril vénérien dans les familles." Par P. Diday, Paris, 1881.

That the gonorrhœal virus and puerperal septicæmia are the great factors in producing the more serious pelvic diseases cannot now be disputed against the weight of evidence; and there can be no doubt that a large number of the apparently puerperal septic cases are of gonorrhœal origin.

Cases Illustrating Phases and Combinations.

Without attempting, at present, to give any complete and precise description of a typical case of gonorrhœal disease in the female sex, including complications, as a guide to diagnosis and treatment, I shall mention in somewhat general terms a few fairly typical cases, with the view of bringing out in concrete form some of the various phases of the disease under consideration, keeping in mind only that, for the present, the disease has to be shown to be of common occurrence, and important apart from its frequency, and leaving definite conclusions as to diagnostic points and treatment to be developed in the sequel.

Case 1.—This patient when first seen was a married woman of 20. She had been married twelve months, and had not become pregnant. She complained of a pain chiefly felt in the left inguinal region, shooting across the hypogastrium, and making her feel sick. She has been worse for the last five or six weeks. Menstruation has so far been regular, but the last two periods have been scanty. Before marriage, menstruation was almost painless, and she was free from inter-menstrual discharge. Now the pain at the periods, coming on a day or two before and lasting until the flow ceases, is intense. It doubles her up, as she puts it; and she cannot sleep at night for the pain. Her ailment began three months after marriage. She had then some sort of acute abdominal disease, accompanied with severe hypogastric pain and scalding in micturition. This illness made her keep to bed for a fortnight. There is also mastalgia in the right side, intensified before the periods. Examination per vaginam shows that the uterus is in normal position and movable. Both ovaries are made out enlarged and adherent, and very sensitive to touch. These notes, taken twelve months ago, indicate the patient's condition then; she is now no better. She has been an in-patient of a hospital for women, and has been subjected to various measures of treat-

ment, with only temporary benefit. She is now a dysmenorrhœal invalid, and in all probability permanently sterile. Here then is a young, robust girl, whose health is deteriorated, perhaps entirely ruined, by marriage. What is the essential element in the causation of this result? I have not been able to detect the gonococcus, but the husband confesses to having contracted gonorrhœa within a year before his marriage, although he considered himself well when the marriage took place. Note the change in the type of menstruation in this case, often one of the most striking features in creeping gonorrhœa.

CASE 2.—The next patient was, at the time of her marriage, three years ago, a robust healthy woman of 22, without any record of illness since childhood. A few months after marriage she began to suffer from vaginal discharge, with frequent and painful micturition. Menstruation, which used to be regular, normal in amount, and painless, became painful and profuse, ultimately developing into menorrhagia, for which medical treatment had to be sought. The first marked menorrhagic period was ushered in by an illness requiring rest in bed, the chief symptom of which was severe hypogastric pain. From this time the periods varied from one to three weeks, and were accompanied with severe pains, which continued during the whole time of the menstrual flow. An intermenstrual discharge is described as "running away" from her. The uterus, it appears, was dilated and curetted by the medical practitioner who first attended her, and the result was temporary relief from the hæmorrhage, but with no relief from pain at the menstrual periods. When she came under my care, a year and a half ago, I found that she had an enlarged uterus, evidently the result of metritis, and both her ovaries were enlarged and painful. She was submitted to treatment, varied in every way that promised to give relief, but with only temporary advantage. The third time she was admitted as an in-patient of the Manchester Southern hospital was after great suffering at a menstrual period, and when she had made up her mind to undergo any operation that might be considered necessary. The state of the ovaries was worse than ever, and accordingly, with the consent of all concerned, the ovaries and tubes were removed. That is six months ago. The tubes were somewhat dilated, and the outer extremities were incorporated with the enlarged and cystic ovaries. The

patient recovered without any bad symptom, and is now relieved of her sufferings, and she has regained to a large extent her healthy appearance.

There can be no doubt, I think, that the direct cause of all this injury to the woman's health was due to gonorrhœal infection: The husband had the disorder the year before he married, and though his statements have varied, he admits to having seen traces of the disease within six months of his marriage. He was and is addicted to drink, and, that being the case, it is easy to understand how the diseased process would be continued indefinitely in the latent form, and be ultimately stimulated by frequent sexual intercourse into such active development as to make it readily contagious.

CASE 3.—In this case we shall see how the diseased process may continue indefinitely, while the woman is living under comparatively favorable circumstances, producing symptoms which, under other conditions, might call for operative treatment. The patient is 29 years of age, and has been married the second time five years. She came first under my care eight years ago, when her first husband was living. At present the form her ailment assumes is enlargement of the uterus, with retroversion and a tendency to prolapse. On exertion, the dragging of the uterus, when not supported, produces nausea and vomiting, which may come on quite suddenly during any kind of active exercise. The left ovary is enlarged and tender, and bands can be made out behind the broad ligament, which are the result of inflammatory adhesions. Near the vaginal orifice there are tender areas, which make it almost impossible for the patient to wear a Hodge's pessary, and yet a padded Hodge is the instrument which gives most relief to the other symptoms. This difficulty seems to be due to a chronic inflammation of the glands of Bartholini. The patient has been so far sterile since her second marriage; she menstruates regularly, but with severe pain, and scanty menstrual discharge. There is a considerable, though not profuse, leucorrhœa. Now, if a physician were being consulted by this patient for the first time, in her present condition, he would probably find it impossible to make out for certain the state of parts or the cause of the disease. It would be possible to elicit the history of a very severe puerperal illness over seven years ago; that illness was of gonor-

rhœal origin. I attended the patient during her pregnancy, while she was suffering from an acute attack of gonorrhœa, conveyed to her by her husband, in whom it was clearly of post-nuptial origin. She was only apparently cured by the end of pregnancy. She was delivered of a healthy child, but she was seriously ill for months. The severer symptoms came on nearly a month after the birth of the child, and arose from inflammation, extending to the pelvic peritoneum and including the ovaries. There followed pelvic abscess, and all the usual train of hectic and emaciation, with adhesions and fixation of the uterus. She made a very slow recovery, and was unfit for any exertion for many months. I have a continuous history of her ailments since then, because I have seen her at intervals during these seven years, and there can be no doubt but that all her troubles began with, and are the direct result of, the gonorrhœal infection during her first and only pregnancy. Since her second marriage this patient's ailment has been "latent." Her husband has never suffered from any sort of irritation of the glands or urethra, hence it may be confidently asserted that the wife's leucorrhœa has been entirely non-specific in its effects, although specific in origin.

CASE 4.—M. N., æt. 22, single, strong, healthy-looking woman, complains of a pain in the left inguinal region, and in the thigh. Menstruation is irregular and profuse, sometimes lasting three weeks, and producing extreme weakness. There is intense dysmenorrhœa, the pain continuing during the first two or three days of the period. Much inter-menstrual discharge. On examination per vaginam, the uterus is found to be enlarged and fixed, with patulous os; there is a large, hard, not sensitive mass behind the uterus, with the usual accompanying hardness of the tissues round the left side of the pelvis. This patient had an illegitimate child twelve months ago, and she has never been well since. In the latter half of her pregnancy she suffered from swelling of the nymphæ and scalding in micturition, and had an extremely profuse mattery discharge. She is convinced it was a venereal disease that she had contracted, although she does not assign the reasons for this belief. The child began to have an inflammation in one eye, with mattery discharge, two days after its birth, and this continued for about a month, although the child was under treatment.

This is a common type of hospital case, occurring among women of the same class, both married and single. Repeated examinations of the discharge for the gonococcus gave no result, but the history permits of a confident diagnosis.

CASE 5.—M. E. M., æt. 24, married six years, and has had four children. Last confinement six weeks ago. Has always had good recoveries until this time. Was confined to bed for three weeks, with pelvic pain after her confinement, and has continued to suffer from the pain to some extent since she began to go about. She has been troubled with a profuse yellow discharge since the lochia ceased. On examination, the uterus is found to be enlarged, with open os (sub-involution), and the left ovary is much enlarged and very tender to touch. Discharge from os, yellow, mattery, profuse. For the last two months of her pregnancy she had a slight discharge, which caused some irritation about the vulva, and a slight discomfort in passing water. She volunteered a statement which left no doubt that her husband was under treatment for a venereal disease contracted during the wife's last pregnancy. The child was under treatment as an out-patient of the Southern Hospital for *ophthalmia neonatorum.* Examination of the uterine discharge in this case, the matter being taken from the os uteri for the purpose, showed the presence of a micro-organism, having all the characteristic appearances of the gonococcus.

CASE 6.—This patient was a rather scrofulous girl, of the domestic servant class, aged 19, who, when first seen, had been suffering for about six months from an ailment which was ascertained to be gonorrhœal in its origin. She had the usual hypertrophied uterus, with great enlargement and pain of the ovaries; the condition of the Fallopian tubes could not be differentiated. Medical non-manipulative treatment and rest produced much improvement in the course of six weeks, but even then the patient could not walk erect. After other six weeks' treatment by medicated vaginal tampons, without further improvement, I resolved to make a careful exploration of the pelvis with the help of an anæsthetic, and then decide on the question of operation. The careful examination was made, a sound was used, which had been thoroughly disinfected—which was, in fact, lifted out of a warm antiseptic solution in order to be introduced—and the result of the ex-

amination was a decision to operate if a short trial of the then universally favorite hot douche failed to give relief. The hot douche was used for the first time the same day, in the hope of relieving the discomfort caused by the examination, and next day the temperature began to rise. This was one of my early cases of the kind, and I temporised in the hope of giving relief by sedatives; but the after course was continuously downward. There was pelvic peritonitis with fixation of the uterus, and intense pain on touch. She never menstruated again. Ultimately pelvic abscesses formed, one bursting into the bowel and another into the bladder, and continued for months to refill and discharge alternately. The patient slowly emaciated, and ultimately died of exhaustion.

My belief with regard to what happened in this case, in the light of later experience, is this: The examination by the sound produced a congestion by its stimulating effect, which, assisted by the hot douche, stirred up into more active development the quiescent gonococci, resulting in a further spread of the inflammatory process, probably producing pyo-salpinx, or abscess of the ovary, and other abscesses. It is just possible that the sound may have introduced some pathogenic organism, which brought on a "mixed infection," or the tube may have been stimulated into discharging some of its contents into the pelvic cavity. The one thing certain about the case, whatever it may have been towards the end, was that it was gonorrhœal in its origin. If the ovaries and tubes had been removed when the temperature began to rise, the result might have been very different.

CASE 7.—Another domestic servant, in whose case the only ascertainable cause for the lighting up of a fresh attack of ovaritis was passing the sound in examination. She confessed to having become affected with a venereal disease, with ordinary symptoms of a mild gonorrhœa, twelve months before. Since then her menstrual periods had become painful, and so profuse that her mistress suspected an abortion at the time I was first asked to see the patient. The girl was so reduced in strength by reason of her losses of blood, and pain from enlarged and tender ovaries, that she was unfit for work. She was admitted, at the request of her mistress, into the Southern Hospital. In the course of examination, a thoroughly disinfected sound was employed, and in 24 hours there was a dis-

tinct rise of temperature with unusual pain in the pelvis. The left ovary became more tender and larger, but in a few days it could not be defined because of a hard exudation in the left side of the pelvis. There was high temperature, with the usual red, dry tongue, but there was never any hectic. The temperature fell in ten days from the first onset of pyrexia, and the exudation could be pressed upon without giving rise to much complaint. All seemed to be going on well for about three days, when the temperature began to rise again, and the right side of the pelvis developed the same train of phenomena as the left. No abscess ever formed, and the patient ultimately recovered after an illness of two months' duration. When last seen her menorrhagia was cured, for menstruation had become scanty, but the dysmenorrhœa continued; she was still unfit for prolonged exertion of any kind.

CASE 8.—Mrs. B., æt. 29, married seven years, had one miscarriage in the first year of married life, no pregnancy since. She came under my care two years ago, and was under treatment over twelve months without deriving any benefit. She had been always strong and healthy before she was married, and her husband is a well-developed, healthy man. On his side there is the history of gonorrhœa contracted about two years before marriage, but there is no evidence of a gleet continuing after the first apparent cure, and there are no habits of dissipation to make such a continuance or recurrence probable. There is no clear evidence of the conveyance of the disorder to the wife, all her early symptoms being possibly attributable to pregnancy. She aborted at four months without apparent cause, and suffered for a long time from some form of puerperal illness, accompanied with pain in the abdomen. No abscess appears to have formed. From this time menstruation became painful and profuse, and at the time she first came under my care she was entirely laid aside during her periods, suffering what was described as an agony of pain. She appears either to have had at these times short faints or some sort of slight epileptic seizures. She was tolerably well in the intervals. The ovaries were enlarged and painful, and there were some obscure points in the palpation, suggesting adhesions. Dilatation of the uterus before a period gave no relief to speak of. The final resort was oöphorectomy. Both the tubes were closed and adherent at the outer ends,

the right being greatly distended with turbid serum. The ovaries contained blood-cysts, and were adherent. The omentum had become attached to the extremity of the right Fallopian tube. Tubes and ovaries were completely removed. The operation took place nearly twelve months ago. The patient made a perfect recovery. She is now well, except for some nervous symptoms characteristic of the menopause.

This case, taken by itself, would prove nothing with regard to gonorrhœal infection. It requires the experience of many other cases more or less closely resembling it to bring out the fact that it is a very good typical case, and that we are justified in attaching what might otherwise seem undue importance to the points which make for proof of its gonorrhœal origin.

CASE 9.—A lady, now 30 years of age, was married twelve years ago, and during the first year of wedlock contracted gonorrhœa from her husband. She had a miscarriage at seven months, about a year after marriage, and since then she has been a wreck. At present she suffers from enlargement of the uterus, which becomes completely anteverted at times on exertion, making even a moderate amount of walking impossible. Her ovaries are enlarged, especially the right, and they become very painful at the menstrual periods. Menstruation is scanty, and very painful. There is now hardly any leucorrhœa. The anteversion of the uterus causes much bladder distress at times. She is anæmic and thin, the reverse of what appears to have been her condition before marriage. There is in this case the history of a long serious puerperal illness, necessitating constant medical treatment for over twelve months. The patient has been under treatment for relapses at short intervals almost continuously since recovery from the first serious attack. She has never become pregnant again. She appears to have worn all manner of pessaries, and to have tried internal and external applications of every kind. She has had the uterus dilated once, resulting in a severe illness of some kind; and she used to have several days of suffering every time the sound was passed by any one of the numerous specialists whom she has consulted in the course of years. Among other therapeutic measures that have been tried has been median incision of the cervix uteri, the only remaining objective evidence of which seems to be a small notch on the posterior lip of the os. What the anatomical

condition is at present and how much of the deviation from the normal is to be ascribed to the original gonorrhœa, and how much to treatment, nobody could now profess to decide. Twelve months ago she consulted, at my suggestion, a celebrated London gynæcologist, who wrote to me: "She seems to me to have very little the matter," and went on to say that, in his opinion, she was suffering more from treatment than anything else, and he had advised her to give it all up, "to walk and dance and enjoy life." She was not satisfied with the advice given her by this physician, because she believed herself too ill to attempt to follow it, but she took his prescription to a chemist to be dispensed. The dispenser entered into conversation with her, and at his suggestion she went the same day to another specialist in the neighborhood whose name she had never heard of till then. This young gentleman wrote to me that he had diagnosed double pyo-salpinx, and had recommended operation. He went on to say: "If you do not care to undertake the operation, I would be *very pleased* to do it. I have been very fortunate with my cases of abdominal section, and *I look upon this as a very favorable case.*" I am responsible for the italics. Out of respect for the feelings of earnest and upright gynæcologists, I shall quote no more of this letter, but I must strenuously protest against any one professing gynæcology, whatever his excuse for ignorance, calling a case with a history of ten or twelve years of complications "a very favorable case" for operation. Favorable it could not possibly be, regarding either the immediate consequences of the operation or the remote results. Such cases are always difficult and dangerous because of the firmness and extent of organized adhesions; and they are disappointing in their later results, because the reorganization of the innervation of the parts is either very slow in following a radical operation, or it remains incomplete. Palliation, not cure, is the best that can reasonably be expected. Enough has been said to call attention, in passing, to these extremes of treatment for some of the complications arising from gonorrhœal infection, and to call up in imagination the effects upon the patient of such advice. In the case under consideration the patient, wisely, I think, has adopted as much as she could of the advice "to give it all up," and is certainly no worse, probably a little better, than she was last year.

CASE 10.—Two years ago I had occasion to open a mammary abscess in a hospital patient, and I took some of the matter to the Pathological Laboratory at Owens College with the object of trying some cultivation experiments, which I had the opportunity of making under Professor Dreschfeld's friendly instruction and guidance. The pus was full of micro-organisms, some of which looked like gonococci. Tube cultivations produced a development like that obtained by Bumm from the gonococcus. No pure cultivation was carried out with this specimen. When the patient was next seen she was asked about her symptoms during pregnancy, and it then came out that she must have been infected with gonorrhœa while pregnant. The infant at the breast was suffering from *ophthalmia neonatorum,* and was also an out-patient of the hospital. The patient had so far no symptoms suggesting the existence of any pelvic complication. Her only ailment was the mammary abscess, which must have been produced by the extension of the inflammation along the milk ducts, and that inflammation must have originated in the implantation on the nipple of colonies of gonococci from the child's eyes. To go back a step further, the purulent discharge from the child's eyes originated in infection from the genital tract of the mother. The state of matters here described may have been a result of "mixed infection;" but without going into the discussion of that subject, I may claim, provisionally, this case as also an illustration of the consequences of gonorrhœa.

Cases might be multiplied indefinitely, but sufficient have perhaps been given for the present object, which is to convey a general impression of the importance and the frequency of occurrence of this class of disorders. It may seem strange to some to whom such observations are the mere commonplaces of practice, that so much is said about the importance of these disorders as a group of diseased conditions. But there can be no doubt that the subject is, as a rule, treated too lightly. It will be seen that a point common to all the cases cited is the absence of treatment in the early stages of the disease. The only apparent exception is the case which was under my care while in the acute stage; but even in this case the disease was thoroughly established before treatment was begun, and, besides, the treatment was of the ordinary inefficient routine sort which was in vogue eight years ago. How little success

attended it has been made manifest. As a rule, the disorder in women excites so little attention that the doctor is called in only to deal with complications when they arise.

The reasons for this are probably the absence in most cases of severe urinary troubles; the want of an evil conscience, which is usually present with the man; the comparative difficulty with which women can minutely inspect their pudenda when slight discomfort begins, and their natural shyness about submitting to the inspection of other eyes; and the comparative frequency with which some plausible reason for a discharge or discomfort can be assigned; it is, for example, the effects of intercourse soon after marriage, a derangement of menstruation from some supposed "cold," or some one of the disorders of early pregnancy. No doubt one active influence in preventing the detection of gonorrhœa in the woman, while it is yet in the curable stage, is the conviction on the part of the husband that the thing is of no importance if the wife can be kept in ignorance of its nature. Matters will assume a very different aspect when all concerned awaken to the importance of prevention, and the difficulty of cure when the disease has reached the stage of complications. It is necessary to guard against exaggeration, for there is a danger that in rousing from long ignorance and neglect of the subject, the professional mind may sway to the other extreme, and, amidst the phenomena of disease obscure in their nature and as yet inexplicable, be tempted to accept gonorrhœal infection as an easy and sufficient explanation of morbid processes with which it has no kind of causal relation. Still, as yet, the actual facts with regard to opinion are all the other way, and the danger which exists is not too strongly stated by a recent writer on the subject, who says[1]:—"The danger of gonorrhœa in the woman lies especially in this fact, that the infection from the urethra or vulva, vagina or cervix, may extend over the mucous membrane of the uterus, and thence further on to the tubes, the ovaries, and peritoneum, where it may give rise to a series of the gravest abdominal ailments. These may end fatally in acute peritonitis, resulting from the escape of pus from the tubes into the peritoneal cavity, or from the bursting of an abscess of the ovary, or of a pyo-salpinx; or they may give

[1] Dr. Fritz Levy (Copenhagen): "Om den 'latente' Gonorre hos Kvinden." *Hospitals Tidende*, Nos. 1, 2 and 3, 1887.

rise to a condition of chronic peritonitis with adhesions of the uterus, tubes, and ovaries to the adjacent organs, or to recurrent attacks of pelvic peritonitis with the ultimate development of a 'gonorrhœal cachexia,' and general breakdown of the health."

FREQUENCY OF OCCURRENCE OF GONORRHŒAL AFFECTIONS.

I have not seen any statistics worth quoting which could guide us to a definite conclusion as to the frequency with which gonorrhœa is to be met with in men and women in this country. Every medical practitioner in the large centres of population knows that it is one of the commonest ailments among men, but with regard to women the case is different. The disease, as it occurs in the female sex, varies so much in its features, that from the signs and symptoms alone it is usually looked upon as impossible of diagnosis. As the cases are not definitely diagnosed, there appears to be a general belief that the disorder is not nearly so common among women outside the classes of professed or clandestine prostitution as it is among men. We must, therefore, for the present, rest satisfied with little more than looking at the views of some foreign authorities, keeping in mind that most of the facts are of foreign origin, when we come to draw conclusions from them.

Noeggerath,[1] whose small work on "Latent Gonorrhœa in the Female Sex" marks the beginning of a new era in our clinical knowledge of the disease, says: "I do not know what the state of matters is in other cities; I did not know how we stood in New York until I questioned the husband of every woman who came under treatment. And I believe we may apply here the dictum of Ricord that in every 1,000 men, 800 have had gonorrhœa." In another place he says: "I believe I do not go beyond the mark when I assert that gonorrhœa in 90 per cent. of the cases remains uncured. Of every hundred women who have married men formerly affected with gonorrhœa, scarcely ten remain healthy, the others suffer from some of the ailments which it is the object of my essay to describe." Coming more into detail with regard to these affections, he says that his experience in New York, which he supposes to

[1] "Die latente Gonorrhœ in weiblichen Geschlecht." Bonn, 1872.

be a type of all the cities of the first rank, leads him to conclude that "after catarrh, chronic perimetritis is the most common ailment of the female sex."

Alfred Fournier, who writes the article "Blennorrhagie," in the *Nouveau Dictionnaire de Médecine*, 1866, says: "Blennorrhagia is perhaps the most common of all diseases. In the great centres of population, in the cities, where the people are facile in their habits and fond of pleasure, there are few men who escape it, and there are many who have had it again and again." In another place he says: "La blennorrhée est vulgairement connue sous les noms de *goutte militaire*, de *goutte matinale*, de *suintement habituel* ou *chronique*. Elle est d'une excessive fréquence, et cela à tous les âges, dans toutes les classes de la société."

Some German gynæcologists have noted the frequency with which the diseases of gonorrhœal origin occur in their practice, and the results strongly support the once much derided statements of Nœggerath.

E. Schwarz,[1] of Halle, in a lecture published last year, says that in 617 cases, carefully observed since the end of 1885, there were 112 which excited the suspicion of previous gonorrhœal infection, and in which the patients' statements and the still existing symptoms made the diagnosis more or less probable. Of these 112 there were 33=5.3 per cent. who proved to be the subjects of acute gonorrhœa, and of that number 19 were either unmarried or widows. In the whole of these cases, diagnosed by appearances and symptoms alone, the diagnosis was further demonstrated by the presence of the gonococcus. Of the remaining 79 cases the gonococcus was found in its characteristic groups in 44 cases, although in some it had to be searched for several times. Thus gonorrhœa was proved to be present in 77 out of 617 cases, that is, in 12.4 per cent. Schwarz is of opinion that many of the remaining 35 cases had been infected at some time or other, and that the gonococcus would have been found if examination had been made at the most favorable times (Prädilektionszeiten), shortly before or after menstruation, during pregnancy, or in the puerperium. Several of them suffered from perimetritic affections with fixation and dislocation of the uterus, purulent

[1] "Die gonorrhoische Infektion beim Weibe."—*Sammlung klinischer Vorträge*. Leipzig, 1886.

endometritis, or showed other suspicious signs, and with several there was a history of purulent ophthalmia in their new-born infants.

Sänger,[1] of Leipsic, who is perhaps the principal champion in Germany of views approaching to those of Noeggerath, gave the latest results of his observations in a paper read before the German Gynæcological Society at Munich last year. He does not consider that the diagnosis of gonorrhœa is established except by the clear statements of the man or woman, by the occurrence of ophthalmia in the new-born children, or by the presence of affections of the urinary tract and the vulvar glands. The last especially he considers pathognomonic. Out of 1,930 gynæcological cases which came under his notice, in private and hospital practice, in the previous year, Sänger found 230 cases of gonorrhœal disease, equal to 12 per cent., or one-eighth of all the cases. More recently still, of 161 cases he had found 29, or 18 per cent., of gonorrhœal origin. In 389 cases of pregnant women, he found that 100 had a purulent discharge, 26 per cent., and 40 of the children ultimately born suffered from ophthalmia neonatorum.

O. Oppenheimer,[2] of Heidelberg, working upon the material of Professor Kehrer's Clinic, endeavored to establish the relative frequency of gonorrhœal infection in women, partly by direct examination of the vaginal discharges, and partly by the occurrence of ophthalmia neonatorum. In 108 pregnant women, he found 30 in whose vaginal secretion the specific diplococcus was undoubtedly present; that is, in 27.7 per cent. Ophthalmia neonatorum occurred in from 12 to 13 per cent. of the cases.

Lomer,[3] of Berlin, examined some of the patients of Schröder's Clinic, just as they came, without any selection on the ground of previously existing catarrh of the uterus, and he found in nine out of 32 lying-in women the characteristic diplococcus enclosed in pus cells, that is, in 28 per cent. The cases in which diplococci in colonies were observed, leading to the suspicion

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[1] "Ueber die Beziehungen der gonorrhoischen Infektion zu Puerperalerkrankungen."— *Verhandlung der deutschen Gesellschaft für Gynäkologie*, Leipzig, 1886.

[2] "Untersuchungen über den Gonococcus (Neisser)."—*Archiv für Gynäkologie*, Bd. XXV., Hft. 1.

[3] *Deutsche med. Wochenschrift*, No. 43, 1885.

of gonorrhœal infection, amounted to 56 per cent. of all examined.

The frequency of the occurrence of the disorder varies so much in different localities that it is, and perhaps always will be, impossible to formulate any statement which will be of general application even to cities. A curious example of this diversity is quoted by Noeggerath in a recent paper.[1] He refers to Winckel's statement that when he was at Dresden he was never able to demonstrate a single fatal case of acute gonorrhœal salpingitis in 400 female bodies examined, whereas soon after his transference to Munich he met with many cases of acute gonorrhœal salpingitis and peritonitis, and among them one fatal case. "In Dresden in eleven years not a single fatal case, in Munich in three years and three quarters three cases."

With regard to the material on which my experience in Manchester has been acquired, I am not prepared to hazard any definite statistical statement at present. In private practice the occurrence of some of the gonorrhœal diseases of women, now that they are diagnosed as a rule, is common enough; but they are by no means the most ordinary ailments which one is called upon to treat. In the current list of patients the disease is never without two or three unequivocal witnesses to its existence. In hospital practice, among the class of women who are not very particular in their notions of personal cleanliness, it is a very common disease. Married men of the operative class, when out of work, appear to contract some form of venereal disease in amazingly large proportion, and to take little or no precaution to save their wives from the misery resulting from the contagion. It is a social question which it would be hard to settle, whether the poorer married women of the working class suffer most from the effects, including sterility, produced by the venereal diseases conveyed to them by dissolute husbands, or from the constantly recurring child-bearing and lactation, which fill up the burdensome prime of life for the healthy wives of the more virtuous or fortunate. Among the purely gynæcological cases, as they occur in my hospital practice, the proportion of cases of gonorrhœal origin is about 1 in 15. Of the cases of pelvic

[1] "Ueber latente und chronische Gonorrhœ beim weiblichen Geschlecht."—*Deutsche med. Wochenschrift*, Dec. 8, 1887.

peritonitis, in any of its forms or phases, gonorrhœa is the cause in an overwhelming proportion, the fraction of the whole next in amount to the gonorrhœal being that made up by neglected cases of abortion.

From the contemplation of such facts as these it is a relief to turn to the other extreme. Milton,[1] speaking of a small town in Cumberland, says: "Having had the opportunity of tracing this disease for many years back, in what was then a very small town in the North of England, where gonorrhœa is now rather firmly established owing to the growth of the place, I was able, by means of the books of a successive line of surgeons, kept for a long time, to make out pretty certainly that in their practice it had, till about forty years ago, only been very rarely seen and sometimes not heard of for years together. All the inquiry I could make tended to fortify this opinion: the general experience seemed to be that gonorrhœa always died out soon after it was acquired." In another place he says:- "During a four years' apprenticeship to a surgeon, who, though living in a very small town, had one of the largest practices in Cumberland, I saw but very few cases of gonorrhœa, certainly not a dozen, though every instance of such a disease must have come to my knowledge. Of these I know that some were caught from sources foreign to the place."

With regard to the prevalence of these disorders, we can readily infer that between their frequency of occurrence in the cosmopolitan cities and dense centres of population, and their rarity in sparsely populated rural districts, there may be every grade of proportional frequency. Consequently, if the profession as a whole is to arrive at a just comprehension of the facts, the conclusions reached by each individual as the result of his experience must be modified and corrected by the experience of those practising under every degree of difference in circumstances. If this fact were kept in mind, we should find the history of the discussion of the subject under consideration less embellished with sneers and triumphant rejoinders than it is. For example, we find Milton discussing Noeggerath's conclusions without having acquired any clearer notion of them than could be obtained second-hand from a review, and with mildly sarcastic superiority brushing them aside. "It does

[1] "On the Pathology and Treatment of Gonorrhœa." By J. L. Milton, 5th Edition, London, 1883.

not seem to have struck Dr. Noeggerath," he says, "that, had his facts been correct, *gonorrhœa would have long ago depopulated every country into which it penetrated.*"[1] It will be seen, however, on reference to Noeggerath's words, which I have been careful to give literally, that he explicitly states that his allegations do not apply to the state of any community in respect of this matter except New York, with which alone he is familiar. If we had only our crowded world-cities to trust to for the continuance of the race, the world would soon be depopulated, or worse, from more potent causes than gonorrhœa. If the statements on either side of the question of frequency of occurrence are a little strong, the conclusion of the whole matter only amounts to this: that for the physician in the large city, gonorrhœa is a disease of primary, practical importance; for his contemporary in the rural district it is more a matter of speculative scientific interest.

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[1] *Op. cit.*, p. 53.

CHAPTER II.

HISTORICAL RETROSPECT OF VIEWS ON THE PATHOLOGY OF GONORRHŒA IN WOMEN.

In order to obtain a clear view of our present position with regard to the pathology of gonorrhœa and the effects of the disorder, it seems to me expedient to give a short historical retrospect of the path by which our present position has been reached. We shall then be better able, without the need for repetitions, definitely and concisely to consider the special phases of the disease with which we are at present concerned; to examine the phenomena of the affection as it occurs in the female sex, the consequences which infection may entail, and the prophylaxis and treatment, and any medico-legal, moral, and social questions which may naturally arise out of the whole case.

For our present purpose the past may most conveniently be divided into three periods—(1) Before Noeggerath's treatise appeared; (2) Noeggerath's work and immediate influence; (3) Neisser's discovery.

Before Noeggerath.

(1) The first period takes us back, according to the ordinary fashion in medical histories, to the time of Hippocrates. For practical purposes we may rest satisfied with looking to the more modern epoch when Ricord flourished. Ricord's views as to the non-specific nature of gonorrhœa dominated the profession in his time, and do to a large extent still prevail. There was always a strong minority who held the opinion that the disease was specific, but they were in the position of mere agnostics, who could not accept the "frivolous anecdotes" on which their opponents seemed to ground their faith, rather than a school of scientific opinion founded on research and observation. The successive failures of the specificists to

demonstrate the presence of an organic virus in the form of a micro-organism was claimed as still stronger support of the antagonistic views. Several investigators, each in turn claimed to have discovered the specific organism. Donné's *trichomonas vaginalis*, for example, held the field for a considerable time as the organism whose presence produced the phenomena of vaginitis; but the anticipations of its discoverer and those who accepted his opinions were doomed to disappointment. But the existence of a micro-organism to carry the disorder was not a necessary part of the belief of those who held to the specific nature of gonorrhœa. The position taken up by Sigmund and Auspitz of Vienna, Durkee in America, and perhaps also by Milton and other authorities in this country, was that gonorrhœa can develop only from gonorrhœa, and some of these certainly held to the view that the specific virus resided in pus as compared with any other form of secretion. On this opinion depended some important practical conclusions, and we shall see that with regard to the precautions to be taken to prevent the spread of the contagion the believers in specificity were as faulty as the opposite camp.

Ricord, Fournier, and Diday were the chief authorities from whom orthodox writers, in this country and elsewhere, drew inspiration, and confidence in the correctness of their theoretic views.

Mr. Henry Lee, in the article in Holmes's "System of Surgery" already referred to, under "Causes of Blennorrhagia," mentions, in addition to the usual direct contagion, the action of irritating substances and constitutional causes. He then, in support of his position, quotes from Ricord a passage which I transcribe as well adapted to our present purpose:—"If we investigate with the greatest care the exciting causes of gonorrhœa—and I am now speaking of characteristic cases of the disease—we cannot help admitting that a gonorrhœal virus is absent in the majority of cases. Nothing is more common than to find women who have occasioned gonorrhœa unsurpassed in intensity and persistency, and attended by the most serious complications, and who are yet only affected with uterine catarrh, which is sometimes hardly purulent. In many cases, intercourse during the menstrual period appears to be the only cause of the disease; while in a large number we can discover nothing, unless, perhaps, errors in diet, fatigue, exces-

sive sexual intercourse, the use of certain drinks, as beer, or of certain articles of food, as asparagus. Hence the frequent belief of patients, which is very often correct, that they have contracted their gonorrhœa from a perfectly sound woman. I am most assuredly familiar with all the sources of error in such investigations, and I will presume to say that no one is more guarded than I am against the various forms of deceit which are strewn in the path of the observer; yet I confidently maintain the following proposition: *Gonorrhœa often arises from intercourse with women who themselves have not the disease.* Any one who studies gonorrhœa without preconceived notions, is forced to admit that it often originates from the same causes that give rise to inflammation of other mucous membranes."

When the masters in this peculiar field of science felt the need of strong language to bolster up such flabby opinions on causation as this passage implies, we need hardly feel surprise at any vagaries of the ordinary disciple. After reading a series of the stories by which the non-specific view is maintained, one hardly feels the absurdity of the suggestion made by a surgeon, mentioned by Milton, that a certain case of gonorrhœa might have originated from making water in the night air.

With regard to the phenomena of the disease in women, the predecessors of Noeggerath held the erroneous view that the vagina was the chief seat of pathological change. If the urethra and bladder were not involved, producing the characteristic symptoms, and if no coarse changes had occurred in the vagina, the surgeon was not in a position to decide with confidence whether or not he had to deal with a venereal disease. A discharge from the uterus was to him the product of a uterine catarrh, a condition quite separate and distinct from any venereal affection, and in only a very few severe cases had the observer confidence to allege any causal relation between the venereal virus and the uterine or pelvic disease. It followed from such loose notions as these with regard to the chief seat of the malady, its superficiality in an anatomical sense, and its non-specific nature, that the surgeon felt himself justified in pronouncing, from mere inspection, any suspected female clean or otherwise. It requires little imagination to grasp the vast social importance of this mis-

taken view of the pathology of so common a disorder; and that it was a mistaken view it is the chief object of this essay to emphasise, because all the rest depends upon it. If a concrete example of the social results of a pathological error be demanded, it should be remembered that the Contagious Diseases Acts, now repealed, were passed and administered in the United Kingdom, partly on the basis of this discredited pathology. Yet the medical profession is so indifferent to revising its opinions on this subject, and so confident in its old prepossessions, that it is probably not going beyond the mark to say that, if the re-enactment of these statutes depended upon a *plébiscite* of the qualified practitioners in this country, taken without further discussion, the laws would at once again come into force.

With regard to the disease, as affecting the male sex, the *crux* of the position was the belief that the pus cell was the pathological unit. The pus was the morbific agent, but inasmuch as any pus cell, whatever its origin, might be sufficient, the disease was not specific. The practical result of this belief was that men, just recovering from gonorrhœa, were assured that if only the remaining discharge was clear and glassy they might indulge in sexual intercourse without fear of conveying the contagion. With some it was a difficult question to decide when the discharge became sufficiently clear to be innocuous, and, for safety, many surgeons stuck to the rule that there must be an entire disappearance of the discharge before they could sanction sexual intercourse. "So long as *any discharge exists* sexual congress is unsafe,"[1] is a dictum containing the most advanced opinion of this school. This expresses by no means the general opinion and practice. "But not only does the medical world believe that a so-called cured gonorrhœa is actually cured, but they are even of opinion that a man who has a gleet (Nachtripper) may not infect his wife. It is usual at the present time with the best-informed practitioners (non-specialists) to sanction marriage in the cases of men who still continue to observe adhesion of the urethral orifice and staining of the linen, as *beaux restes* of a gonorrhœa. Even the highest authorities, as Professor A. Geigel, permit the cohabitation with his newly-married

[1] Mr. Henry Lee, in Holmes's "System of Surgery."

wife of a man, the subject of gleet, so soon as the urethral discharge appears perfectly clear."[1] Such was the state of practice and of doctrine with regard to gonorrhœa, when a specialist in diseases of women began to comprehend and explain the meaning of certain pathological conditions of the female sexual organs which he had long observed. The establishment of the relation of cause and effect between the neglected, apparently trifling and innocuous, chronic gleet in the male, and inflammation of the pelvic peritoneum in the female sex, is one of the most important and interesting chapters in modern gynæcology.

Noeggerath's Work and its Immediate Influence.

(2) The work which I am specially desirous of doing justice to, because of its originality and intrinsic merits, is that of Dr. Emil Noeggerath, a German physician, formerly practising in New York. His "grand idea" is expounded in a treatise, a mere "opuscule" in respect of physical proportions, published in German in 1872. The peculiar mode of publication has been a misfortune to English readers, and it has largely detracted from the influence which the book ought to have exercised. It has never been translated into English, and this fact seems to justify me in drawing largely upon the words of the original in support of the views which I am seeking to advocate. When it first began to dawn upon me in the course of gynæcological practice that many of the unexpected mishaps, and the whole course of many of the most intractable cases, were due to the gonorrhœal virus, I naturally turned to the literature in order to learn what was already known on the subject. Comparatively late in the search I had the opportunity of reading Noeggerath's monograph, which, until then, I had known only by hearsay. Like every other man I have hitherto met, I knew of Noeggerath as a German doctor who had actually published the outrageous doctrine that clap is an incurable disease, and as nothing more. It was, therefore, surprising to find, on perusal of the work, that only the undeniable exaggerations had been seized upon and noised abroad; the admirably clear expositions of the fruits of clinical work of the highest excellence, and the great idea

[1] Noeggerath, p. 2.

which inspired the whole and formed an advance on all that had hitherto been written on the subject, appeared to have fallen, for the most part, as seed on stony ground.

Noeggerath's work was largely clinical, and in this respect it contrasts with much that is to follow, which is mainly the work of pathologists and bacteriologists, engaged in research and observation without such close relation to the actual phenomena of disease. He, however, clearly enunciated the microbiotic nature of the disease, and indeed he almost believed, like some of his predecessors and contemporaries of the specific school, that he had discovered and succeeded in cultivating the special organism. "There is hitherto awanting," he says, "a so-called direct proof. If it were already proved in which form-elements the gonorrhœal contagion is exclusively bound up, if it were demonstrated that the pus corpuscles, and the pus corpuscles only, were the carriers of the contagion, then perhaps elements might be discovered in the secretions of the woman by careful microscopic examination which would throw light upon the origin of the affection.". . . "The whole meaning and importance of micrococci and of the fungi derived therefrom bear as yet so clearly the stamp of incompleteness, and are viewed with distrust by so many distinguished men of science, that I cannot expect any of my readers to attach very great importance to the presence of a fungus in forming their conclusions on the question. The subject must be further investigated by the mycologists in co-operation with one or other of the gynæcologists." Although it would be an exaggeration to call upon the reader to admire these sentences as a prophecy from the remote past, or as bearing the evidence of far-reaching scientific vision, still they show a very clear appreciation of the state of contemporary knowledge, and of the course that future research must take in order to be successful. It is evident that if Noeggerath had enjoyed the advantage of knowing Koch's methods of investigating the life-history of micro-organisms, that the missing link in the chain of evidence which he sought for would soon have been found, and the discovery of Neisser might have been anticipated by seven years at least.

Let us now examine Noeggerath's clinical work. He says that his object in publishing his observations is "to depict in their characteristic features a group of diseased conditions

peculiar to the female sex, and to prove that certain phenomena observed in the sexual organs, which have hitherto been looked upon and treated as distinct, possess a common basis from which, collectively and separately, they are derived." For this common basis he selected the name "latent gonorrhœa," because "it may be present for weeks, months, and even years, in the affected individual before it displays itself by any of its characteristic phenomena." In explaining further the nature of latent gonorrhœa, he says: "I have undertaken to show that the wife of every man, who at any time of his life before marriage has had gonorrhœa, with very few exceptions, becomes affected with latent gonorrhœa, which sooner or later shows its existence by some one of the forms of disease about to be described. The reason why this fact has not hitherto been brought under discussion in a radical fashion is grounded on several circumstances. In the first place, we physicians have hitherto believed that a gonorrhœa in a man, after it has once ceased to exhibit any signs of its existence, such as occasional moistening or adhesion of the urethral orifice, even itching, or discomfort in micturition, is actually cured. Such is, however, in the great majority of cases, not the fact, and I believe I am not going beyond the mark when I say that 90 per cent. of cases of gonorrhœa remain uncured. Of 100 women who have become the wives of men who have formerly been affected with gonorrhœa, scarcely 10 remain healthy; the rest suffer from one of the ailments which it is the task of this treatise to describe."

In order to prove his theses, Noeggerath gives the notes of fifty selected cases, which are so arranged as to illustrate the various heads under which he discusses his subject. These clinical groups, under which he classifies his observations, are: (1) Acute perimetritis; (2) recurrent perimetritis; (3) chronic perimetritis; and (4) ovaritis. All the four varieties of diseased processes are complicated with catarrh of distinct portions of the mucous membrane of the sexual organs, and this catarrh, in a few cases, is found to be the only sign of disease. Although this classification may now be considered obsolete, if even it could be considered originally established by the author's own material, it did good service by calling attention to the extreme frequency of perimetritis as a consequence of the gonorrhœal infection. It may be a question whether it is

possible clinically to distinguish between the three varieties of perimetritis, or whether every attack of perimetritis is not necessarily always more or less acute, ultimately becoming more or less gradually chronic, and liable at any time, under favoring conditions to become recurrent. In any case we may accept that which is implied in the classification as something which clinical experience since Noeggerath's time tends more and more to establish, viz., that there is always catarrh, and in the great majority of cases perimetritis. "Inflammation of the female sexual organs, as it occurs in consequence of latent gonorrhœa, whether acute or chronic in its course, is in every case periuterine—that is to say, it affects the serous covering of the pelvis, and of the organs contained in it." In 100 cases of this disease, occurring in the author's practice, not one can be recalled to memory in which the morbid process began in the parametrium. After a criticism of the views of Matthews Duncan and the statistics of Bernutz, he concludes that, provisionally setting aside the cases which are said to have originated in the puerperal state and from menstruation, Bernutz also supports the opinion that gonorrhœa and syphilis are the most prominent causes of perimetritis. "But not only does the florid, acute gonorrhœa supply the principal contingent of this class of disease, but the so-called latent gonorrhœa is quite as important as the acute form. And I may add (1) that a woman who, at any time of her life, has had an acute gonorrhœa, has to expect some time, it may be after months or years, one or more attacks of acute, sub-acute, or chronic perimetritis; (2) that the wife of a man who, at any time of his life, before or during wedlock, has suffered from gonorrhœa is, with regard to an attack of perimetritis, in the same position as if she herself had had an acute gonorrhœa

"Under these two groups of circumstances a woman must expect at some time or other in her life to become the subject of a pelvic inflammation, sometimes ending only at death. If it comes to an acute manifestation of the affection, the case usually takes something like the following course:

"The man, either already a dweller in a large city, or in his capacity as traveller, student, etc., has become initiated into the mysteries of modern civilisation by contracting gonorrhœa. This disease, although it may have apparently disappeared without leaving a trace behind it, makes him for many

a day capable of conveying the contagion. The young, hitherto healthy, wife begins to complain a few weeks after marriage; attention to her domestic duties becomes a burden to her, and pedestrian exercise which could formerly be taken without the least effort now gives rise to signs of fatigue; menstruation becomes more profuse than formerly, and there are pelvic pains during the first days of the periods; a little vaginal discharge follows each period, and this, gradually increasing, ultimately continues without intermission until the next menstrual period begins. After a few months, really severe pains come on either in the left or the right half of the pelvis, and the sufferer is ultimately compelled, on account of feverishness and unbearable burning in the abdomen, with increased discharge, to take to bed and send for medical help. According to the severity of the attack she remains confined to her bed for weeks or perhaps for many months, with exhausted strength, struggling for life, ultimately slowly recovering, but remaining sterile and invalid for the rest of her days.

"It not unfrequently happens that the woman who is married under such circumstances soon becomes pregnant, and during her pregnancy she suffers from pelvic pains which are supposed by herself and the doctor to be necessary drawbacks of her condition, and so she receives no particular attention. It sometimes happens that the symptoms become so urgent as to call for active treatment in order to prevent miscarriage. Labor ultimately comes on, and is followed by a severe endometritis with perimetritis, the former or the latter being the more prominent feature of the case. The development of this inflammation may begin either immediately after the birth, or, as is more frequently the case, it may come on eight to fourteen days, or even six to eight weeks after."

It is a peculiar fact that some women directly after marriage with a man who has formerly had gonorrhœa begin to exhibit symptoms of serious inflammation, whilst others may be years before they emerge from the latent period, and others again show such trifling departures from the normal condition that it is difficult to say whether they are affected or not. It may be set down as a law of this disease that "the earlier the first cohabitation occurs after the time when the gonorrhœa was pronounced to be cured, the shorter is the period of latency in the woman, and the acuter are the symptoms."

Space will not permit me to transcribe all Noeggerath's interesting cases, and it must suffice to select one from the first series.

CASE II (Noeggerath's Case 5 [1]).—"Mrs. M., a native of Boston, had been, when I first saw her, married five years to an advocate in New York, who about one year before his marriage had undergone two months' treatment for gonorrhœa. The wife, who before marriage was a type of robust health and beauty, began soon after marriage to have ailments; she remained sterile, and suffered from pain at the beginning of her menstrual periods, and, what had never before occurred to her, began to have a slight fluor albus shortly before the periods. After a year she consulted Dr. Marion Sims, who performed on her his operation of incision of the cervix, with the object of curing the sterility. This proceeding was followed by such severe hæmorrhage as to necessitate tamponade of the vagina. On the following days pain began, and gradually increased to an enormous severity. The doctors in attendance found that the cause was an acute perimetritis. The patient was confined to bed for two to three months, and since that time she has never had a day's good health. She consulted one after the other the principal gynæcologists of New York and Boston; by one she was cauterised for ulcer of the cervix; another applied a large blister to the hypogastrium on account of chronic metritis; and a third made her wear an intra-uterine pessary for two months. A careful examination revealed the following facts: the uterus was anteverted and but slightly movable; the left ovary was small, hard, and firm, and fixed in the pelvis by adhesions; whilst the right ovary, lying deeper, seemed to be greatly swollen, rounded, and softened. Both ovaries were intensely painful to touch. The vagina was reddened, and bathed in a muco-purulent discharge, and there was catarrh of the glands of Bartholini. This lady had suffered inexpressibly during the last four years, not only at the menstrual periods, but during the intervals. The sleep was broken. . . . . I gave a very doubtful prognosis, and as soon as I saw that neither narcotics nor absorbent medicines produced the least effect upon her condition, I advised her to visit a German watering-place.

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[1] *Op. cit.*, p. 18.

My advice was concurred in bv a gynæcologist in Paris, and the patient on my strenuous recommendation spent two seasons at Kreuznach. On her return she felt in many respects better, but by no means completely restored to health." This patient ultimately left New York on account of symptoms of commencing tuberculosis, and the author could learn nothing of her subsequent history.

The chapter on "Recurrent Perimetritis" contains some passages which cannot fail to arrest the attention even of those familiar with the literature of the subject. The author has never seen a case of recurrent perimetritis in which he failed to prove the former existence of gonorrhœa either in the woman or in the man. He cannot understand how the phenomena of the disease are to be explained, except by the occasional emptying of the irritating contents of one or other Fallopian tube into the pelvic cavity, but as to the reason why this accident should occur in only a few women he cannot offer any suggestion. He thinks that any stimulus capable of producing sudden powerful contractions of the uterus and Fallopian tubes may produce in that way expulsion of the contents of the tubes into the pelvic cavity, and among these causes, which have been illustrated by cases of specific tubal catarrh in his own practice, he mentions: coitus after long absence of the husband, introduction of the uterine sound even in cautious and experienced hands, the application of bland intra-uterine medicaments, the wearing of intra-uterine pessaries, incision of the external or of the internal os, dilatation by sponge tents, and other manipulations. And such results he has observed only as the consequence of manipulations in women who were the subjects of latent gonorrhœa. "At least the presence of this affection explains, in by far the largest number of cases, the occurrence of the so-called 'accidents,' which, to the astonishment of the much-cutting uterine surgeon, sometimes come upon him like a thunderbolt from a cloudless sky. . . . I am far from saying that every fatal result of these minor operations is the result of perimetritis due to tubal catarrh, and I am persuaded that phlebitis and lymphangitis supply their contingent. This last, however, is the exception, the first the rule."

The notes of a case of recurrent perimetritis make, as a rule, a long story, and it would be tedious if I were to quote any of

Noeggerath's cases in full. We are concerned at present only with the relations of supposed gonorrhœal infection to the after-history, and the author's evidence in proof of such relations can be exemplified by extracts from a few typical cases.

His eighth case is that of Mrs. F., a well-developed and beautiful Englishwoman, who was married to a young merchant in 1861. The gentleman had been under treatment for a rather obstinate gonorrhœa by a friend of Noeggerath's, but for three months before his marriage not a trace of the disease was to be seen. He was assured that it was quite safe for him to get married, and he acted on the advice. All went well for about six weeks, and then the lady began to complain of a pain in the side, which at last increased to such a pitch that she had to take to bed. The husband's doctor was consulted, and diagnosed pelvic peritonitis, which he treated energetically. The patient was confined to bed for two months, and has been ailing ever since—1861 to 1872. All the symptoms are referable to the condition of the pelvic organs, which is given in detail. Then follows the history of ten years of bad health, varied with periods of more acute suffering.

The next case is that of a lady who was brought to New York in the beginning of 1872 to be under the author's care. When a bachelor the husband had suffered from gonorrhœa, which required many months for its cure; but it had disappeared for two years before marriage. In answer to the question whether every trace of it had disappeared, he admitted having noticed, after his marriage, an adhesion of the urethral orifice, and to have experienced occasional discomfort in micturition. Ten months after marriage the wife was confined of a healthy child, and since then (18 years) she has never become pregnant again. Soon after her confinement she began to complain of pain in the left side, and a sense of weakness in the pelvis. Then follows a history of wanderings in search of health from one European health-resort to another, and back to America. There were six distinct attacks of pelvic inflammation. Digital examination proved the existence of a mass of exudation in the pelvis, enlargement of the ovaries, and matting together of the pelvic organs generally. Noeggerath evidently despaired of doing any good, and recommended her to visit a Canadian watering-place.

These are fair examples of the half-dozen cases given as illustrations in this portion of the work. They have all these two points in common, that they are incurable ailments, entirely destroying the health and happiness of women who were healthy before marriage, and that they all show a history of gonorrhœa in the husband, which is brought out in a more or less convincing fashion as the cause of the wife's sufferings.

One more case may be quoted, chiefly for the sake of illustrating a point to which attention must be called later on, viz., the import of *neglected* gonorrhœa in men in relation to the gonorrhœal infection in women.

CASE 12 (Noeggerath's Case 19).—"Mrs. V., an American lady, married six years before, as a healthy girl, to a man who suffered from a neglected gleet until shortly before marriage. The husband assured me that for two months before marriage he was quite well. Directly after the marriage the wife began to complain, had a leucorrhœal discharge, and suffered from pain in the right side, and sacralgia. Menstruation also became irregular and painful. She was treated by a series of doctors without any lasting benefit. On examination, I found a very sensitive 'succulent' uterus dislocated to the left, both ovaries very painful, especially the right, which was enlarged and fixed close to the uterus in an exudation mass. There were large ulcerated areas on the labia, and catarrh of the glands of Bartholini. After two months' treatment the patient had a slight attack of acute perimetritis." . . .

In the chapter on chronic ovaritis and catarrh, the author calls attention to the importance of inflammation of the glands of Bartholini as a sign of previously existing gonorrhœa. It has been usual to rely on the presence of an inflammation of the urethral mucous membrane as a diagnostic mark, but this is comparatively evanescent; the reddening of the orifices of the vulvo-vaginal glands, on the contrary, remains for years. "In the natural condition the openings of the ducts of both glands are seldom visible to the naked eye. When, however, you examine a woman who in former years has suffered from gonorrhœa, it is the rule that if you pull the labium minus away from the remains of the hymen, you see, between the upper and lower caruncula myrtiformis, a deep red point, covered with the glassy mucus. From this point, as a centre, there extends upwards and outwards a streak, which grad-

ually becomes paler, and ultimately fades into the color of the surrounding mucous membrane." These appearances are said to be characteristic of catarrh of the glands of Bartholini. That such an inflammation is caused by gonorrhœa, and that it is persistent, there can be no doubt, but whether it is characteristic of gonorrhœal inflammation alone, or may result from inflammatory conditions set up by other causes, yet remains to be settled by further clinical observation.

On the subject of sterility as a consequence of latent gonorrhœa, Noeggerath carries his conclusions further than the evidence produced seems to justify. He maintains that gonorrhœa is a common cause of sterility in the man, producing azoospermia, and he gives cases in proof. He is more convincing in dealing with the analogous effect on women, and he makes out a better case, though here also he leaves an impression of going beyond the record in the statement of opinions.

His treatment calls for little notice, not because of any deficiency in its merits, but because for our present purpose it is irrelevant. It is the treatment pursued by a judicious, scientific, and most patient physician, yet the most striking thing about it, perhaps, is its inefficacy. Noeggerath never hints at operative treatment, except to condemn it, but it is probable that few chapters have ever been written in more eloquent and telling support of surgical interference in this class of ailments than the portion of his short work which is devoted to treatment. Neither is this the place to deal with any conclusions which may suggest themselves from considering Noeggerath's work in the light of later experience, or to discuss wide-reaching questions which arise out of the subject and to which he himself explicitly calls attention. His more definite conclusions are perhaps best given in a nearly literal rendering of his own words:—

(1) "Gonorrhœa in the man, as well as in the woman, persists for the whole life-time, in spite of apparent cure.

(2) "There is a latent gonorrhœa in the man as well as in the woman.

(3) "Latent gonorrhœa in the man as well as in the woman, may evoke in a hitherto healthy individual either a latent gonorrhœa or the symptoms of an acute attack.

(4) "Latent gonorrhœa in the woman manifests itself in

course of time by perimetritis, acute, chronic, or recurring; or by ovaritis, or as a catarrh of some definite portions of the genital mucous membrane.

(5) "The wives of men who at any time of their lives have had gonorrhœa are, as a rule, sterile.

(6) "Such women, if they do become pregnant, either abort or bear only one child. Exceptionally three or four children are born.

(7) "From the discharge of a woman affected with latent gonorrhœa a fungus may be cultivated, which is exactly analogous to that obtained from the discharge of acute gonorrhœa in the man."

Much of this is now merely matter of historic interest; as the expression of opinion which first attracted serious attention to the subject, and led to the further observations which have brought about an entire revolution in the professional estimation of the importance of this class of disease.

Noeggerath has lived to greatly modify his first impressions. In the paper already referred to (p. 22) he states that he has given up his pessimistic stand-point, viz., that gonorrhœa is incurable, and that ninety per cent. of all married men who have had this disease infect their wives. He admits that if you ask ten men, eight or nine may say they have had gonorrhœa, yet it would be unjustifiable and arbitrary to assume that any disease of the organs of generation occurring at a later time in the wife is due to gonorrhœic infection.

When husbands deny that they have retained traces of gleet or a gonorrhœa until just before marriage, Noeggerath's rule is to thoroughly examine the urethra. Some of the worst cases of acute and recurring perimetritis which he has seen have occurred in the wives of husbands who had been dismissed as cured from gonorrhœa shortly before marriage. Of 24 patients suffering from acute and recurring perimetritis 12 were married to men who were cured one to three months before marriage, one man had been cured 5½ months, another a year, six had been healthy two years before marriage, two married 2½ years after stoppage of the gonorrhœa, one had had no symptoms of disease for 10 years, and one for 11 years. These are the opinions formed by 15 years' further experience. His views with regard to puerperal infection will be mentioned in the proper place.

Noeggerath's first work was more an influence than an extensive addition to the sum of knowledge; a fruitful idea, under whose inspiration other men saw in a fresh light the facts of daily experience, and could recognize in them the material suitable for building up the required superstructure of new knowledge. We can only look at the work of the most important contributors, those who by observation and research have added the largest amounts to our knowledge, or who have modified in some important respect the general views by the contribution of influential ideas.

The first fruits of Noeggerath's work in Britain appeared early, if the crop has not been extensive since. In 1873 Dr. Angus Macdonald[1] read a paper before the Obstetrical Society of Edinburgh, which contained a report of some striking cases in support of Noeggerath's position, and an expression of opinions which might almost be called prophetic. In the course of his remarks he says that Noeggerath "really seems to prove his positions well, strong and strange though they appear to be. I must confess, however, that I cannot help feeling convinced that he proves too much. . . . That, however, Dr. Noeggerath has got hold of a grand idea, and one which possesses a large amount of evidence in its favor, no one, I think, can deny who reads the recorded cases fairly." The author then proceeds to give the details of seven cases, in which gonorrhœal infection had given rise to more or less severe puerperal illness. The gonorrhœal puerperal fever was in one sad case fatal. The whole of Dr. Macdonald's cases go to disprove a point that Sänger has recently insisted upon, viz., the constantly late onset of the gonorrhœal form of puerperal fever as compared with the early appearance of symptoms in cases originating otherwise. That some gonorrhœal puerperal cases are late in developing their severer symptoms, there is little room for doubt, but the cause of variation in this respect is still in obscurity. For the sake of calling attention to this point, which is obviously important, and interesting inasmuch as it is still unexplained, it may be worth while to insert here Dr. Macdonald's first case, and

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[1] "Latent Gonorrhœa in the Female Sex, with Special Relation to the Puerperal State." By Dr. Angus Macdonald.—*Edinburgh Medical Journal*, June, 1873.

place by it for comparison one which came recently under my observation.

CASE 13 (Dr. Angus Macdonald's Case 1).—"Mrs. S., aged 24, was delivered of a female child after an easy labor, on the 15th of April, 1869. Previous labors two, and had been quite normal. Both mother and child did well until about the fourth or fifth day, when the mother was seized with severe pain in the abdomen, accompanied with great tenderness on pressure, and a considerable amount of fever. This pain and tenderness, however, disappeared in about a week or ten days, under the influence of opiates internally, and the application of poultices externally. About the same time as the mother became ill, the baby's eyes were noticed to be affected with severe conjunctivitis of a suspiciously specific character. On inquiry, I ascertained that the husband of my patient had, during the time she was carrying this child, been affected with discharges of a gonorrhœal character, but that she herself had not suffered in consequence, except that she had some leucorrhœa before her confinement. She had had no pain on micturition, nor any swelling of the parts, as far as she could remember. The child's eyes were treated." . . . The mother made a slow recovery, but was able to be out of bed by the end of five weeks. "About this time, after being at church getting her child baptized, my patient was suddenly seized with excruciating pain in the left side of the pelvis, about the situation of the left ovary, accompanied with quick pulse and fever. This, on physical examination, gave all the usual signs of a perimetritis originating near the opening of the left Fallopian tube, and spreading forwards and towards the right, so as to involve the anterior half of the pelvis, and fix the uterus in position. For this attack she was treated by poultices and opiates, as well as, latterly, with the bromide and iodide of potassium, and other remedial agents. She made an exceedingly slow recovery, and was more than six months almost entirely confined to bed. . . . Since then I have ascertained from her that she suffers more or less severely at each menstruation from pain in the left groin, and that when she menstruates she is usually ill for nearly three weeks at a time, though not very much blood comes away at any one time. Since I attended her, in 1869, she has given birth to two healthy children. . . . She is now in tolerably good health."

CASE 14.—B. J., æt. 24, married two years and a half. Had one child a year after marriage. She came under my care owing to a chronic ailment, which was ascertained to be perimetritis, with double laceration of the cervix uteri. The anamnesis supplied the following facts:—The patient was well until her confinement; she had copious leucorrhœa during her pregnancy, but no urinary trouble. Two weeks after her confinement some severe inflammatory illness, affecting the abdomen, came on, and she was in bed several weeks. She has suffered from pain in the left iliac and inguinal regions more or less ever since. The child suffered from an inflammation of the eyes, with discharge of matter, for which it was successfully treated by the doctor in attendance on the mother. There have been several relapses since the patient's recovery from the first acute illness, the last dating from a fortnight before she came under my observation. In an interview with the husband, I learned that he had a gleet for over a year before his marriage, but considered himself quite cured for two months at least before the marriage took place. He observed, however, for a long time, that after sexual intercourse there used to be a moistening and adhesion of the meatus. This patient is still under treatment, and other facts of the case may have to be referred to in illustration of points which would at present seem irrelevant.

There can be no doubt that the case is one of late gonorrhœal puerperal fever, and the course of the disease is that of a recurrent perimetritis. This is a fairly typical case, and the present purpose is served by placing it in juxtaposition to Dr. Angus Macdonald's typical case of early development of the inflammatory symptoms. But the relation of gonorrhœal infection to the puerperal diseases is too important to be treated only incidentally.

The late Dr. Thorburn, of Manchester, paid some attention to gonorrhœa in its relation to gynæcology, but soon concluded that it was unimportant. At the meeting of the British Medical Association held at Manchester in 1877, he read a paper on "Latent Gonorrhœa as an Impediment to Marriage," of which a too concise summary is given in the *British Medical Journal*, of August 25th, 1877. Dr. Thorburn denied the possibility of Noeggerath's conclusions being correct, and "appealed to the statistics of 81 private families, carefully

collected by him. He showed that there had been 33 per cent. of male gonorrhœic infections previous to marriage, 26 in all; and taking all the cases of abortion, sterility, uterine, and pelvic inflammations, and living births that had occurred in these eighty-one families, he showed conclusively that there had been the merest fractional difference in their proportion between the previously and not previously infected classes. As regards inflammatory pelvic affections, the balance was fractional in favor of the free gonorrhœic cases; in other respects equally fractional in favor of the non-gonorrhœic." The conclusion was that the latent gonorrhœa of Noeggerath is mythical; and is not, as it otherwise would be, an imperative barrier to marriage. It is not for me to go behind the statements here quoted and try to explain them away or minimise them. They do not seriously affect the question whether or not gonorrhœa may be an important factor in the causation of the diseases with which the gynæcologist has to deal. It must at once strike the reader, possessed of local knowledge, that Dr. Thorburn's statistics would be drawn from a class of people, exceptional to some extent in this respect that they would be specially careful and cleanly in their habits, and specially careful of their own health and that of their families, reducing to the minimum both the chances of contagion and the consequences of contagion, if perchance it did occur. However, I am concerned at present only in giving illustrations of the clinical work done under the influence of Noeggerath's "grand idea," and before the discovery of the gonococcus. We come now to the work of Neisser.

Neisser's Discovery and its Influence on Research and Observation.

So early as 1869, Hallier[1] had discovered and described the cocci which occur in gonorrhœal pus, but in the absence of sufficient means of magnifying, coloring, and illuminating the objects examined, his work attracted little attention.

In 1879, Dr. Albert Neisser,[2] at that time assistant in the

[1] *Zeitschrift für Parasitenkunde*, 1869. Bd. I., p. 179.

[2] "Ueber eine der Gonorrhoe eigentümliche Micrococcusform."—*Centralblatt für die medicinischen Wissenschaften*, 1879, No. 28.

University Clinic for Dermatology at Breslau, published an account of his observations. Whatever suggestions may have been thrown out by previous investigators, there can be no gainsaying the fact that it was Neisser's publication which gave the stimulus and guidance to workers in the same field, and has led to an ever-increasing and widening process of research and experiment, by both clinicians and biologists.

Neisser found that when he prepared the pus of gonorrhœa in a certain way, he could see, in and about the corpuscles, micrococci, which he concluded must be characteristic of the disease. He put the thinnest possible speck of pus upon the object glass, and allowed it to dry; he then stained the pus with a watery solution of methyl violet, and allowed it to dry once more. It was then ready for examination. In his investigations, Neisser used a Zeiss's microscope with Abbé's illuminating apparatus, $\frac{1}{12}$th oil immersion lens, with ocular No. 4 or 5. On bringing the object into focus, besides the nuclei of the pus cells, varying in form and deeply stained, while the protoplasm took on the dye but slightly, there could be seen more or less numerous groups of micrococci. Single individuals were seldom to be seen; almost always there were two micrococci so close to each other as to give to the observer the impression that they were one organism. He described each double individual as forming a figure-of-eight, or as a pair of dinner-rolls placed with the flattened surfaces close to each other. He believed that they developed by a process of division, each half of the double organism gradually dividing and forming a fresh double organism. This process is shown in diagrammatic form in the accompanying illustrations taken from Bumm's work (Fig. 2c). The micrococci for the most part formed colonies of 10, 20, or more individuals, and they were surrounded by a mucous envelope, which could be distinctly seen under a more subdued light. The micrococci were found chiefly on the surface of the pus corpuscles, rarely on the epithelial cells. In some pus corpuscles which were beset with micrococci, the nucleus was wanting or diminished in size.

Neisser, at the time of publishing his first essay, had found the micrococcus in 35 cases which he had examined. In every case examined this was the only sort of micro-organism found in the gonorrhœal pus, with the exception of a case which was suspected from the first of being complicated with soft chancre

of the urethra. On the other hand, this particular micro-organism was not present in the pus from every other form of venereal disease or complication. Nor could it be discovered in any one of thirteen cases of simple fluor vaginalis submitted to examination without selection.

Exactly the same typical micrococci of gonorrhœa in the male were found in nine cases of purulent urethritis in women. In the seven cases of purulent ophthalmia of the new-born, which had so far come under examination, the same characteristic micrococcus was found in great abundance. In all cases of simple conjunctivitis, which were examined for the sake of comparison, the micro-organism was looked for in vain. It was present in the only two cases of gonorrhœal ophthalmia in the adult which had come under observation.

The presence of the micrococcus consequently appeared to Neisser to be a constant mark of a gonorrhœal affection of the urethra, and of the eye; and even at the time of publishing his observations, he had repeatedly made use of this fact in order to diagnose the specific gonorrhœal character of pus.

The publication of this discovery in Germany, at a time when Koch's countrymen were full of confident expectation as to the future of the new bacteriology, attracted to it universal attention, and brought a host of workers into the field. It was not to be expected that the results of all observers would agree, and the history of seven years' industrious observation and experiment shows a good deal of difference of view on even essential features of the subject. Neisser himself has been constrained to modify his original opinions, but, upon the whole, his work has been only supplemented and established by subsequent observers. The reader to whom the subject is comparatively new, when endeavoring to get at that which is acquired and added knowledge, amid a good deal of conflict of evidence, even in matters of fact, should not be discouraged into relinquishing the quest. He should remember that while most of us look upon Jenner's discovery as one of the greatest boons ever conferred upon mankind by medical science, there are still not a few who denounce it as a curse; and among these there are to be found men who have had the opportunity which a medical education offers of acquiring scientific modes of thinking, and so subordinating their emotions to their intellect when discussing scientific questions. Similarly, it will be

possible, I think, to trace in the work of some contributors to the elucidation of the questions arising from Neisser's discovery, the influence of fixed and rooted opinions, and perhaps also of bias of even less creditable origin, in judging of new facts.

As Neisser's discovery emanated from a clinic for dermatology, the dermatologists and syphilologists were among the first to publish observations. The presence of the micro-organism in the opthalmia of the new-born interested the ophthalmologists and obstetricians, and other inquirers soon followed suit. It seems to be the custom for the specialists in Germany to publish their results, for the most part, in their special journals, and so the literature of a subject such as that under consideration, which claims the attention of several specialists, is almost inaccessible to the foreigner. Bumm, of Würzburg, whose work we must chiefly draw upon, mentions fifty-two contributions up to the beginning of 1886, and I find forty papers on ophthalmia neonatorum alone abstracted in the *Centralblatt für Gynäkologie,* in the six years 1881-86, some of them, like Credé's, being considerable treatises. It is only possible, therefore, in trying to get a clear view of the present state of opinion on all that relates to the gonococcus, to select merely the contributions which have added something to our knowledge and have stood the assay of criticism and later experience.

In 1880, A. Bokai[1] published the results of attempts to cultivate the micro-organism. He succeeded in obtaining pure cultivations, and in two cases he believed that he was successful in producing acute gonorrhœa in men by transferring a few drops of the fluid containing the crop of pure gonococci to the urethra. In the same year F. Weiss,[2] of Nancy, prepared a thesis, under the guidance of Prof. Spillmann, in which he described afresh the organisms observed in gonorrhœal pus, and alleged that it was constantly found in a large series of cases.

Haab,[3] who has published at least two important papers on

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[1] "Ueber das Contagium der acuten Blennorrhoe."—*Allgemein. med. Central Zeitung*, 1880, No. 74.

[2] Weiss: Thèse: Nancy, 1880. Published in *Annales de Dermatologie*, 1881.

[3] Haab. *Korrespondenzblatt für schweizer Aerzte*, 1881, Nos. 3, 4; and same Journal, 1885, Nos. 1, 2.

the ophthalmological aspects of the subject, found the cocci of blennorrhœa neonatorum absolutely identical with those of gonorrhœa. He met with the figure-of-eight coccus constantly in the virulent inflammation of the conjunctiva, and as constantly failed to find it in the pus produced by simple catarrh. In the second paper, which is devoted to the "Etiology and Prophylaxis of the Ophthalmo-blennorrhœa of the New-born," he says that he recognizes the disease as of gonorrhœal origin only when he has made out by the microscope the presence of gonococci. Ordinary vaginal and lochial discharges do not produce blennorrhœa, but a simple purulent catarrh; without the microscope the differential diagnosis is impossible in the early stage.

After several of the ophthalmologists had published their observations, all of them adding to the proof of the justness of Neisser's original statements, Neisser[1] himself again came forward with the results of more mature investigation. He maintained that the gonococcus is a distinct organism, both functionally and morphologically. He again described its size and shape, and explained that its occurrence always in masses or colonies, never in chains, depends upon its mode of development, which is by a process of division at right angles to the long diameter of each element of the double individual. It is absolutely constant in every gonorrhœal inflammation of a mucous membrane, and it occurs in no other disease. He had succeeded in obtaining pure cultivations of the organism.

About the same time an important contribution to the subject was made by Leistikow,[2] assistant in the Clinic for Syphilis in the Berlin Charité Hospital. He failed to find any bacterium characteristic of hard or soft chancre, but he was able to demonstrate the occurrence of Neisser's gonococcus in the gonorrhœal inflammation of all the mucous membranes. He found that the gonococci were not on but inside the pus corpuscle, and that the development of a large number of them caused bursting of the cell-wall. His attempts at inoculating

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[1] "Die Micrococcen der Gonorrhoe."—*Deutsche med. Wochenschrift*, 1882, p. 279.

[2] "Ueber Bakterien bei den venerischen Krankheiten."—*Charite-Annalen*, VII., p. 750. Abstract in *Centralblatt für die med. Wissenschaften*, 1883, No. 22.

various animals with pure cultivations of the gonococci gave no result.

In the following year Bockhart,[1] of Würzburg, published a curious experiment, whose results introduce us for the first time to some acquaintance with the tissue changes produced by the development of the gonococcus. He obtained pure cultivations of the gonococcus, and he injected some of the fourth generation into the hitherto perfectly healthy urethra of a man in the last stage of dementia paralytica. Two days later the external orifice of the urethra was found to be slightly reddened, and a drop or two of a mucous secretion could be expressed. In the course of the two following days the scanty discharge had become purulent. On the fifth day a copious thin mattery discharge could be pressed out from the canal. From the third day onwards microscopic examination revealed the presence in the pus of innumerable gonococci. The patient died at the end of ten days, and thus Bockhart was enabled to make a microscopic examination of the diseased mucous membrane. He concluded that the gonococci are the pathogenic bacteria of the gonorrhœal diseases. When brought into contact with urethral mucous membrane they penetrate, probably between the epithelial cells, into the lymph spaces of the mucosa and submucosa of the fossa navicularis; here they multiply and give rise to active inflammation and migration of white blood corpuscles. They enter the white blood corpuscles, and penetrate with them into the blood vessels and push on in the connective tissue of the mucosa and pars cavernosa upwards towards the bladder. They destroy the white corpuscles into which they have migrated, and are themselves ultimately destroyed in the tissues or in the blood stream. Thus far, observers are unanimous in their agreement with Neisser, but now Sattler and Eklund, working at different subjects, announced their opinion that the micrococcus cannot be specific. The former concluded that he had obtained the characteristic, so-called specific gonococci, in the discharge from the eyes of a new-born child purposely inoculated with the vaginal secretion of an apparently healthy woman. The latter believed that he had found exactly similar

[1] "Beitrag zur Aetiologie und Pathologie des Harnröhrentrippers." —*Sitzungsbericht d. phys. med. Gesellschaft zu Würzburg*, 1883, Nos. 1 and 2. Abstract in *Centralblatt für Gynäkologie*, 1884, No. 36.

and apparently identical micro-organisms in other purulent discharges than those of gonorrhœa. Schirmer,[1] assistant in Sattler's clinic at Erlangen, has also mentioned one case in which typical blennorrhœa of both eyes came on in a new-born child from accidental contact of some vaginal discharge of the mother with one of its eyes. He says the mother was quite clean, but he does not refer to any microscopic examination of her discharges or to any attempt at cultivation of the organism in proof of the truth of his assertion. His evidence, therefore, becomes reduced to the "frivolous anecdote" rank, such as the reported cases in which men are alleged to have contracted specific urethritis from perfectly healthy women.

Of quite a different sort is the evidence of Zweifel,[2] then also of Erlangen, it may be noted. Lochial discharge, varying in time post partum from the 3rd to the 13th day, was taken from lying-in women who certainly had never suffered from gonorrhœa, and applied with certain precautions to the conjunctiva of healthy new-born children. The lochial secretion was first carefully examined microscopically for cocci, and a few cocci but no gonococci were discovered. The experiment was tried in six cases, and not only did no simple ophthalmia or specific blennorrhœa arise, but their eyes remained perfectly sound.

E. Arning,[3] working in Neisser's clinic, discovered the gonococcus in the discharge from the inflamed glands of Bartholini in eight cases of gonorrhœa. He colored the cocci with methyl blue and the cell protoplasm with eosin. He was of opinion that the gonococci were *in* the cells, but he dissented from Bockhart's statement that they penetrated the nuclei.

Amidst a series of papers emanating from various sources, but adding little or nothing to our knowledge, we come upon E. Bumm's[4] first contribution. His subject is gonorrhœa in women, and he announces the discovery, in the vaginal

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[1] "Die Augenentzündung der Neugeborenen."—*Centralblatt für Gynäkologie*, No. 14, 1882.

[2] "Zur Aetiologie der Ophthalmoblennorrhœa neonatorum."—*Archiv für Gynäkologie*, Bd. XXII., Heft 2.

[3] "Ueber das Vorkommen von Gonokokken bei Bartolinitis."—*Vierteljahrsschrift für Dermatologie und Syphilis*. X.

[4] "Beitrag zur Kenntniss der Gonorrhoe der weiblichen Genitalien." —*Archiv für Gynäkologie*, Bd. XXIII., Hft. 3.

secretions, of more than one diplococcus exactly similar in form to Neisser's gonococcus. The real pathogenic organism he could separate from the non-pathogenic only by means of pure cultivation. The substance of this paper is contained in the author's large work, which we have yet to examine, and we may therefore omit any further reference to this early production.

Kammerer[1] announces the discovery of the gonococcus in the fluid contained in the knee-joint in gonorrhœal rheumatism. But the evidence does not seem very conclusive. He mentions two cases, in both of which there was a history of injury to the joint, and the proof of the existence of either rheumatism or gonorrhœa is not satisfactory.

Kroner,[2] of Breslau, read a paper in the gynæcological section at the meeting of the German Natural Science and Medical Association at Magdeburg in 1884, in which he gave the results of observations on 92 cases of the ophthalmia of the new-born. He found gonococci in 63 cases, and in 29 cases he failed to find them, in spite of the most careful search. He therefore concluded that there are two forms of blennorrhœa of the new-born, but apart from the presence or absence of gonococci he cannot make out any clinical difference between the forms. Kroner leaves the theme of his observations without any very definite statement of opinion. The subject has yet to be worked out, and that can only be done where the material of a lying-in hospital and an eye hospital can be brought into comparison and carefully and patiently dealt with.

At the same meeting Sänger,[3] of Leipzig, brought forward the subject of "Gonorrhœal Disease of the Uterine Appendages and its Operative Treatment." He stated that the hope aroused by the discovery of Neisser, that in the gonococcus we should find a means of diagnosing chronic gonorrhœa, had proved to be vain. He held it as an established fact that gonorrhœa could exist without the demonstrable presence of gonococci. The absence of the gonococcus proved nothing against the gonorrhœal nature of the disease, whilst the

[1] "Ueber gonorrhoische Gelenkentzündung."—*Centralblatt für Chirurgie*, No. 4, 1884.

[2] "Zur Aetiologie der Ophthalmoblennorrhœa neonatorum."—*Archiv für Gynäkologie*, Bd. XXV.

[3] *Archiv für Gynäkologie*, XXV. 1.

presence of diplococci, in view of the occurrence of non-pathogenic forms, did not prove the gonorrhœal nature of the disease. If the cocci cannot be found, they may have been somewhere broken up, whilst a ferment produced by them may still be active; or they are absent from the secretion while present in the tissues; or there exists—and this would render the high degree of infectiousness of the comparatively trifling amount of secretion in latent gonorrhœa the most intelligible —a permanent form (Dauerform) of the gonococcus not yet discovered. He suggests, by way of analogy, Koch's discovery of the spores of the bacillus of splenic fever, unknown till then.

This contribution of Sänger's, to which, as well as to the more mature essay already quoted from, reference will have to be made again, seems to me a most important one. From my own experience, I should say that there can be little doubt as to the difficulty of finding the gonococcus in chronic gonorrhœa in women. In cases of only a few months' standing, most certainly gonorrhœal in their nature, which have been under treatment, it has been almost invariably impossible to discover the gonococcus, however numerous other bacterial forms might be. If this be established as a fact by general experience, the sooner it is recognized as a fact the better. Fuller knowledge of the life history of the organism must furnish the explanation, and may lead to notable practical conclusions. The absence of the gonococcus in old standing cases is in striking contrast to its uniform presence in acute or sub-acute cases in the invasion stage. But of this more has to be said in its proper place. It should be remembered, however, that Neisser[1] has found the gonococcus in a remarkably large proportion of old chronic cases in men, and further experience may produce similar results from the gonorrhœal catarrh of the cervix uteri. Westermark's discovery of gonococci in the exudation of salpingitis is not a case in point.

In 1885 we have, besides several papers of practical value, such as Oppenheimer's already referred to, a series of observations by E. Fränkel,[2] which as yet stand almost alone and

[1] "On the Contagiousness of Chronic Gonorrhœa."—*Transactions of the Congress of German Naturalists and Physicians at Strassburg*, 1885. Abstract in *Centralblatt für die med. Wissenschaften*, 1886, No. 32.

[2] "On the Results obtained from the Examination of the Secretion in Colpitis occurring endemically."—*Deutsche med. Wochenschrift*, 1885, No. 2.

unexplained. He found in the discharge from non-gonorrhœal colpitis in children, diplococci which were in every respect absolutely identical with the gonococcus. Fränkel's communication elicited a further contribution on this obscure subject from J. Cséri,[1] of Budapest, which, unfortunately, contains no results of the cultivation experiments referred to as having been instituted. The danger of infection from some cases of colpitis in children has been long well known to the staff of the Stephanie Children's Hospital, in Pesth. Since October, 1883, the discharges in 26 female children, varying in age from two to ten years, had undergone microscopic examination, and in every one a large diplococcus bearing a striking resemblance to Neisser's gonococcus had been found. Biologically both cocci appear to be identical. The contagiousness of the discharge is very great, and contact of it with the conjunctiva may lead to serious results. Cséri mentions the case of a short-sighted nurse, who, in syringing a child with colpitis, got her eye infected by accident, and lost it in consequence of conjunctival blennorrhœa.

Further observations on this subject were published by J. Widmark[2] in the same year. He merely states that he has found the gonococcus in eight cases examined, but he does not seem to have proved the identity of the micrococcus present with Neisser's gonococcus.

The most recent French literature on the subject of the gonococcus is summed up in Martineau's[3] work. The author has worked at the subject alone, and in company with Goguel, and is perfectly satisfied as to the existence and pathogenic nature of the gonococcus. He claims for Bouchard precedence of Neisser, by one year, in the discovery and description of the micro-organism. The observations and experiments made by the French physicians have been the same in kind, though not so extensive as those carried out in Germany, but no one is referred to as having failed to satisfy himself of the importance of Neisser's discovery.

This fact is in marked contrast to the import of the con-

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[1] "On the Etiology of Infectious Vulvo-Vaginitis in Children."—*Weiner med. Wochenschrift*, 1885, Nos. 22 and 23.

[2] "Gonococci in eight cases of vulvo-vaginitis of children."—*Archiv für Kinderheilkunde*, VII.

[3] "Léçons cliniques sur la blennorrhagie chez la femme." Paris, 1885.

tributions from Italy. In that country several workers have submitted the results of their German contemporaries to a destructive criticism, and have sought to prove that the gonorrhœa diplococcus of Neisser, considered as the pathogenic element in blennorrhagia, does not exist.[1] The last of these, Giovannini, merely gives categorically a summary statement of conclusions derived from work in the Pathological Institute of Bologna. Until he has described the method by which his results have been obtained, we may suspend our judgment.

[1] De Amicis: *Revista clinica terapeutica*, March, 1884; and Giovannini: *Centralblatt für die med. Wissenschaften*, No. 28, Nov. 27, 1886.

CHAPTER III.

CONTEMPORARY PATHOLOGY AND THE "GONOCOCCUS-NEISSER."

WE have now passed in review the history of Neisser's discovery, and the chief episodes in the course of the evolution of our knowledge of the subject which have modified the original conclusions with regard to the morphology and functions of the gonococcus. Setting aside in the meantime the objections which have been raised on the basis of more or less complete and mature investigations, and accepting provisionally the broad conclusion that the gonococcus is the active agent, the pathogenic element, in the production of the gonorrhœal diseases, whether they affect the sexual organs of the adult or the conjunctival mucous membrane of the new-born, we may now proceed to look at the organism more closely, and consider how we are to detect its presence and how we may bring our knowledge to bear upon the diagnosis and the treatment of disease. Some refinements with regard to morphologically similar micro-organisms, and the mode of differentiating them from the true pathogenic gonococcus, can be considered when we have become more familiar with the ordinary methods of looking for the bacterium.

In order to save frequent repetitions and references, I ought to state explicitly here that in writing this chapter I have constantly drawn upon the work of Dr. Ernest Bumm, of Würzburg, whose treatise[1] is by far the most important and original that has so far appeared upon the pathology of the gonorrhœal infections.

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[1] "Der Mikro-Organismus der Gonorrhoischen Schleimhaut-Erkrankungen 'Gonococcus-Neisser.'" 2d Edition, Wiesbaden: J. F. Bergmann, 1887.

and apparently identical micro-organisms in other purulent discharges than those of gonorrhœa. Schirmer,[1] assistant in Sattler's clinic at Erlangen, has also mentioned one case in which typical blennorrhœa of both eyes came on in a new-born child from accidental contact of some vaginal discharge of the mother with one of its eyes. He says the mother was quite clean, but he does not refer to any microscopic examination of her discharges or to any attempt at cultivation of the organism in proof of the truth of his assertion. His evidence, therefore, becomes reduced to the "frivolous anecdote" rank, such as the reported cases in which men are alleged to have contracted specific urethritis from perfectly healthy women.

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lamp it will take perhaps a minute to dry. This time may be saved, without drawback to the preparation as far as rapid clinical inspection is concerned, by immersing the wet cover-glass momentarily in absolute alcohol, which should be kept in readiness in a suitable vessel, such as a small wide-mouthed stoppered bottle. When the water is replaced by the alcohol, the drying above the flame of the spirit-lamp takes only a few seconds. A small drop of Canada balsam, dissolved in benzol, is now let fall upon the centre of the cover-glass, which is at once applied to the slide, previously cleaned and warmed. The preparation is now complete and ready for examination. With a little experience of this method, and when the appliances are all held in readiness, the practitioner will find that this method of looking for diplococci takes little more time than the ordinary testing of urine. There are obvious objections to the process if it be looked upon as a method of scientific research, but that is not its object. A quantitative analysis of the sugar in urine by even the shortest process requires time, but a qualitative analysis can be, and has often to be, made in the course of a consultation. So it may be desirable to know, in the course of a consultation, whether a discharge contains diplococci, and if it does, the overwhelming probability will be that the case is gonorrhœal, the anamnesis and the clinical features being available to perfect the diagnosis. For the purpose in view the method of examination which I have described is sufficient.

Bumm, in the monograph already referred to, says that at the Würzburg Clinic for Obstetrics and Gynæcology, they take only two or three minutes to make the preparations. For staining a watery solution of magenta (fuchsin) is used: the secretion is spread *upon the slide* and prepared in nearly the same way as that just described. When it is finally dried, the examination is made without a covering glass direct in the oil for the immersion lens.

Neisser's original method of examination could not be finished in one day because of the slow process of drying and staining, and Eschbaum's rapid method takes twenty minutes or half-an-hour. No process requiring such a length of time for its completion is suitable for the consulting-room, however admirable results it may afford in the pathological laboratory.

If it be desired to retain specimens for repeated examination

and reference, all that is further required in the preparation of them is to employ a saturated solution in aniline water of the dye-material selected, and to take plenty of time in drying.

Now the next question is: What do we see in the examination of an object prepared as described? If there are gonococci in the field they will be distinctly seen. When absolute alcohol is used to promote rapid drying, the staining material may be found to have spread a little into the intercellular spaces, but not so as to interfere appreciably with the efficiency of the process. In Fig. 1 we have an attempt to represent the field of view, in a slide prepared from a real case, that of a young man who had been declared cured of a gonorrhœa a

FIG. 1.

month before he came under examination. He was exactly in the condition in which men used to receive, and perhaps do still receive, medical sanction to marry. In consequence of "taking a glass of beer," he had the slightest possible return of discharge, and a speck of sero-purulent fluid supplied the object which the figure is intended to represent. It will be seen that the leucocytes are for the most part merely stained, and free from diplococci, the neuclei taking on a deeper coloration than the protoplasm. One epithelial cell occurs in the field. The diplococci appear only as black dots, for the most part double, but not uniformly so. Some observers see a good deal more of detail in the individual organisms, but no refine-

ments are to be expected from the rapid mode of preparation of the object. The appearance is, however, characteristic and unmistakable. In addition to the groups in or about the cells, it will be seen that two larger diplococci are visible with the epithelial cell as a background. These have probably fallen away from some disintegrating group; but, whatever be their origin, they are a common enough feature in the field of view in the ordinary course of searching a preparation for the gonococcus.

The illustrations in Fig. 2 are intended to show the relation

FIG. 2.—*a*—This group contains: 1, a normal pus corpuscle; 2, a pus corpuscle partly surrounded with cocci; 3, a pus corpuscle with cocci in the protoplasm; 4, a pus corpuscle completely filled with cocci, and 5, a free colony or group of cocci. *b*—Gonococci from a pure cultivation. *c*—A diagrammatic representation of the development of gonococci. *d*—Section from the conjunctiva, showing the presence of gonococci in the discharge before penetrating the epithelial covering.

of the micro-organisms to the other elements that come into view in the field during examination, and also to show some of the details which should be made out by the observer after employing more careful modes of preparation than that just described. They are taken from Bumm's work, and are, as he explains, mostly diagrammatic. The explanatory notes sufficiently indicate the various relations of the micro-organism to the tissue elements, which may be seen in preparations of gonorrhœal pus.

It may now be asked: If in examining a discharge by the method described, the observer finds diplococci in groups or

colonies, such as those figured above, is he justified in giving a decided diagnosis of gonorrhœa? Unfortunately the subject is not so simple as this, and a definite unassailable conclusion is not to be reached by such a summary process. In a suspected case, the discovery of diplococci arranged in the manner which has been described, makes it almost, but not altogether, certain that the disease is gonorrhœal. How then can we be certain? This brings us to the consideration of the diagnostic value of the presence of the gonococcus, and of a method by which it can be infallibly differentiated from any other micro-organism.

There can be no doubt that Bumm has proved the existence of micro-organisms which cannot be distinguished by the individual form alone from Neisser's gonococcus. He describes five different varieties of diplococci obtained by pure cultivation, only one of which, producing a yellowish-white film in the cultivation medium, is pathogenic. It is obvious that cultivation experiments cannot become a clinical method of differentiating micro-organisms which are identical or very similar in form, and we must therefore look for some more practical method. Such a method appears to have been discovered by Dr. Gabriel Roux,[1] of Paris. It depends upon the effect of Gràm's staining fluid upon micro-organisms in general, including the diplococci resembling the gonococcus, as compared with its effect upon the gonococcus itself. Roux's application of Gram's method of staining consists in first coloring the dried film of secretion which is to undergo examination with methyl-blue or gentian violet, fixing the color, if it is fixable, with Gram's iodo-iodide solution, and then washing with absolute alcohol, so as to decolorise the pus or leucocytes in the secretion. Gram's liquid fixes the color in all micro-organisms, as far as is known at present, excepting the gonococcus. Hence it follows that if the cocci, which have been observed in a specimen of discharge prepared in the ordinary way, completely disappear after the application of Gram's solution and washing with alcohol, they are Neisser's gonococcus. If on undergoing the same treatment they remain visible, that is, retain their color, then they are some other form of diplococcus; and the case from which they are derived cannot be pronounced gonorrhœal on the strength of the microscopic examination alone.

[1] *Le Concours Médical*, Nov. 13, 1886.

Roux's method has been submitted to a severe trial by Dr. Charles W. Allen[1] and Dr. Edmund C. Wendt,[2] of New York, and they both express confidence in it as an exact test. These observers have worked together, and though writing in different journals they practically give identical reports. Dr. Allen says: "We have examined pus containing a great variety of forms of bacteria, and find that, while the double coloration method brings out all other micro-organisms more beautifully than any other process we have employed, decolorisation removes the staining from the gonococci and causes them to disappear, while other micro-organisms are not decolorised. Here, then, we have a method which, when carefully carried out, appears never to fail, and has the great advantage of simplicity." The process of examination, as practised by himself and Dr. Wendt, is thus described: "A drop of pus is spread into a thin layer by pressing between two glass slides and allowed to dry in the air. A drop of a solution of methyl-blue in aniline water is now placed upon it for a moment, and washed off with a stream from a wash-bottle; a few drops of Gram's iodo-iodide liquid is then poured on, and allowed to remain for several minutes. This fixes the color on micro-organisms in general. Gram's liquid is now washed off, and while the specimen is still wet, a cover glass is placed upon it, and it is examined with an oil immersion lens. If micro-organisms resembling the gonococcus are found, we proceed to test them by decolorisation. The cover-glass is removed, and the specimen treated with absolute alcohol, until the color is as completely removed as possible. The cover-glass is replaced, and the specimen again examined, when all gonococci will have been found to have disappeared. All other organisms, however, which may have been present will be distinctly visible. If desirable, the pus cells may be brought out again by applying a solution of eosin. By this method we have been able to exclude all cases which would have been of necessity left doubtful, without some confirming test."

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[1] "Practical Observations on the Gonococcus, and Roux's Method of Confirming its Identity."—*Journal of Cutaneous and Genito-Urinary Diseases*, March, 1887.

[2] "A New Color Test (Roux's) for the detection of the Gonococcus." —*Medical News*, April 23, 1887.

It has yet to be seen whether the experience of other observers will confirm Drs. Allen and Wendt in their confidence in Roux's test. Bumm is satisfied, from his experience of the behavior of the various organisms under staining processes, that the pathogenic diplococcus which he found in puerperal cystitis and in mammary abscess has its color fixed by Gram's staining fluid, while the gonococcus has not. But if Bumm's contention is well founded, there is still room for further evidence with regard to the behavior of the four other non-pathogenic varieties of diplococcus. Practically, in dealing with the discharges from the female genitals every source of error in identifying the gonococcus may be considered eliminated. If in a suspected case the particle of matter to be examined be carelessly taken from the lower portion of the vagina, there is a whole "fauna" of that region usually brought into the field of the microscope. But in every actual case for investigation, during the most characteristic and prolonged stages, the discharge for examination may be best taken direct from the orifice of the urethra or from the os uteri, and the fluid taken from either site is comparatively free from bacteria other than the gonococcus. I have constantly applied Roux's test for the last few months, and though as a test it seems satisfactory, I doubt whether, clinically, its application is not a mere counsel of perfection, only valuable in hushing quibbling and pedantic objections to the sufficiency of the clinical and ordinary microscopic methods of differentiation. Instead of examining the same film of mucus or pus first with the ordinary stain and again after the application of Gram's fluid, I have usually made six complete and permanent preparations from each specimen of discharge to be examined, and have labelled them so that they could be repeatedly compared, if necessary. Three of these preparations are made with the ordinary stain, one rapidly done, the other two more carefully; two others are prepared with Gram's fluid, washing in absolute alcohol, and mounting in Canada balsam as described; and the sixth is re-stained with eosin in order to make the pus and other cells again visible, as recommended by Roux. Without exception, in the cases ascertained to be gonorrhœal by independent evidence, when diplococci have been seen at all, they have disappeared under Roux's process, and this has been so whether the pus has been taken from the cervix uteri and been free from other bacteria, or from the

vagina when the diplococci were associated with myriads of other bacilli and cocci which persisted in the preparation after the washing with absolute alcohol.

In old standing cases of gonorrhœal origin, and in doubtful cases, diplococci have hardly ever been seen in the uterine discharge, and when they have occurred in non-gonorrhœal cases they could never be mistaken for the gonococcus, if the grouping of the gonococcus be characteristic.

With regard to the identification of the gonococcus in the actual clinical work of gynæcology, we are now in a position to collect the points of the complete process. They are:—

(1) The presence of some clinical sign or symptom to suggest the existence of comparatively recent infection.

(2) The collection of the secretion to be examined from the cervix uteri, from the orifice of the urethra, or exceptionally from the orifices of Bartholini's glands. As there is, strictly speaking, no vaginal blennorrhagia, the collection of the discharge from the vagina is merely inviting the risk of error.

(3) The presence of a diplococcus *in* the protoplasm of the pus corpuscles, or other elements of the fluid, and its arrangement in the groups or colonies which have been proved to be characteristic. A single group in the field of the microscope is sufficient to settle the question. In the absence of such a group, however numerous separate diplococci or groups of diplococci apart from the cells may be, we cannot pronounce the case to be gonorrhœal, even though we know that separate individuals and groups are very numerous in the acuter stages.

(4) Roux's test may be applied to complete the proof: (*a*) When all the other conditions of the proof are already fulfilled, in order to observe the disappearance of the gonococci, and (*b*) when some diplococci are present, but not in the characteristic groups, in order to see whether the persistence of the micro-organisms will prove the negative.

Presence of the Gonococcus—Its Clinical Significance.

The whole subject is in too inchoate a state to make it safe to allege that these conditions are sufficient to satisfy the requirements of medico-legal investigation. Clinically, so far as my experience goes, the fulfillment of the first three condi-

tions has justified me in stating that a disease was gonorrhœal when such a possibility was denied by the patient or by one of the parties to its production, and, *without exception*, the confident statement of the opinion has elicited information that was previously withheld. But this is anticipating what we have next to consider, viz., the diagnostic value of the presence of the gonococcus in a discharge. All experience goes to show that the presence of the gonococcus settles the question; it is the pathognomonic sign of the disease. Even the apparent exceptions support this conclusion. *If gonococci are present in the discharge from an inflamed mucous membrane, the discharge is of gonorrhœal origin.* Apparent exceptions to this rule have already been referred to. On the one hand, there are the reported cases of vulvo-vaginitis of young children, in which diplococci were observed, which could not by any means be differentiated from the true gonococcus. The later observations of Cséri, during an epidemic in Budapest, point, however, strongly to the conclusion that the virus of that disease and of the gonorrhœa of adults is identical. On the other hand, we have Kroner's observations on cases of ophthalmia neonatorum, in which the gonococcus could not be found in the discharge. Bumm's experiments, however, prove that the discharge from such cases is not infectious, and the inflammatory process is not followed by the same results as are seen in the genuine infectious ophthalmia of the new-born. Further inoculation experiments by Bumm with the discharge from the cervix uteri left as an effect of a former gonorrhœa, and with other discharges of gonorrhœal origin, but free from gonococci, the experiments of Zweifel with the vaginal discharges of puerperal women upon the eyes of the new-born, and a host of other experiments with every variety of discharge, lead us inevitably to the further conclusion that *a secretion containing gonococci, when brought into contact with a mucous membrane capable of infection, gives rise with certainty to a gonorrhœal inflammation;* and conversely, *a secretion, whatever its origin may be, which does not contain gonococci, is incapable of giving rise to a gonorrhœal inflammation.* The portion of the proof of these propositions, which consists of inoculation experiments with gonococci, derived from pure cultivations, is worthy of more detailed statement. The successful inoculations on the human

subject have been, from the nature of the case, very few; attempts to inoculate the lower animals have been uniformly unsuccessful. Gonorrhœa seems to be a purely human development. We have seen under what circumstances Bockhart's inoculation experiment was carried out; let us examine one of Bumm's more satisfactory experiments.

The original virus was obtained from a case of blennorrhœa neonatorum, and the inoculation was effected with gonococci of the second generation of pure cultivation in blood-serum. A speck from the cultivation area, along with a little clot of the serum, was conveyed by means of the platinum wire into the urethra of a woman, measures having been taken to prevent the spread of the disease, should it arise, to the internal sexual organs. The greatest care had been taken to ascertain that the genitals of the subject were entirely free from gonococci. Two days passed after the inoculation without any symptoms. On the third day the patient complained of a burning pain in micturition, and a small amount of yellowish watery secretion could be pressed from the urethra. This discharge contained separate gonococci as well as groups upon the epithelium. The other numerous pus corpuscles present were quite free from cocci. Next day the urethral mucous membrane, which had become red and swollen, bulged out from the orifice. The secretion was now thin, purulent, and small in quantity, and it contained numerous free cocci, epithelium cells beset with cocci, and also some pus cells filled with them. The further course of the disease was that of a rather severe attack of urethral gonorrhœa. The acute stage lasted three weeks, and the discharge required other three weeks of treatment to bring it to a complete cessation. No complications occurred. The discharge was regularly examined during the whole course of the attack, and the presence of the gonococcus in the characteristic manner was as regularly demonstrated. An almost exactly similar experiment was made with the twentieth generation of a pure cultivation of the gonococcus with a similar result.

These inoculation experiments of Bumm's fulfill all the requirements of a complete proof that the micro-organism is the cause of the disease. We have in them (1) a distinct type of disease in gonorrhœa; (2) the isolation of the organism developed in that disease, and its cultivation in artificial

media apart from the tissues in which it originally developed; (3) the inoculation of the organism into tissues capable of taking on the diseased action, and the production thereby of a disease characterised by all the phenomena of the original disease; and (4) the identification of the micro-organism in its characteristic relation to the tissues undergoing the diseased process set up as the result of inoculation.

If the proof, as stated so far, has done justice to the available material for proof which exists, we should now be in a position to accept the dictum—*without the gonococcus there is no gonorrhœa;* and, conversely—*without gonorrhœa there is no gonococcus.* The truth of these propositions is far-reaching in its influence. Gonorrhœa is the only disease which produces an infectious catarrh of the female genitals. All the various other diseased conditions and irritations producing catarrh of the genital tract in the female, which have been alleged to give rise to urethritis in the male, must be admitted to be innocent; they cannot produce a specific blennorrhœa. What then, it will be asked, becomes of the cases with which we are all familiar? We have all seen men who have had the alleged misfortune to contract a urethral inflammation from their wives; they have indulged too imprudently in sexual intercourse; the wife has a chronic discharge from general bad health, or from the mechanical irritation of a pessary; she had not quite ceased menstruating, and so forth. It is an ancient and convenient superstition, which has been almost universally accepted as fact. For such facts so much the worse if they do not square with the well-established modern doctrine. That a simple urethritis, *simulating gonorrhœa,* is not possible, it would be illogical and absurd to allege; but that few of us ever see such a urethritis, whose origin is above suspicion, the universal experience must admit to be the fact. When we consider, on the one hand, how rare alleged simple urethritis is, and how common the specific form; and, on the other hand, how frequently, one might almost say universally, some amount of uterine or vaginal catarrh is found among parous woman, who have never run the risk of specific catarrh, we must admit that there exists a discrepancy which is inexplicable on any hitherto accepted theory. If simple urethritis in the male could be produced by any form of non-specific catarrh in the female, then urethritis would be one of the

commonest of ailments among continent married men, just the class among whom it is seldom or never seen. No case of alleged simple urethritis need, however, cause more than a momentary difficulty. Such cases are always seen in the acute stage; if the discharge contains gonococci, it is specific; if it is free from gonococci, the inflammation is not gonorrhœal. This facility for learning the truth may be sometimes embarrassing to some patients, and occasionally to the practitioner; but the existence of an exact criterion in the early stage cannot fail, in the long run, to conduce to the morality and to the physical well-being of the community.

A case which well illustrates these points has recently been under my care. It was that of a lad of sixteen, a member of a highly-respectable family, who was brought to me by his mother. He had recently come home from school, and he had not been able to conceal the fact that he was suffering from a discharge from the penis. On examination I found that he was the subject of marked phimosis, and the orifice of the urethra could not be brought into view. The purulent discharge from the opening of the prepuce was copious enough. Not so long ago I might have been satisfied and silenced by the explanation which was given me as to the cause of the disorder. But I made a microscopic examination of the pus, and found typical gonococci. The possibility of my view, as stated to the patient alone, being correct was at first strenuously denied, but while he was under treatment I ascertained the origin of the malady. There was nothing unusual about it.

The occurrence of ophthalmia in a new-born child should also lead to an examination of the discharge. If the matter contains gonococci, the disease is gonorrhœal, and in the absence of proof of post-partum infection, it is to be presumed to have originated in gonorrhœal infection of the mother. This is now a universally accepted opinion, and the facts calls for no further notice.

Development of the Gonococcus in the Tissues—Its Effects.

The immediate dependence of the phenomena of the gonorrhœal inflammation upon the presence of the gonococcus

brings us to this conclusion, that the pathology and pathological anatomy of this disease are described in any satisfactory account of the life-history of the gonococcus and the changes which its development produces in the tissues which harbor it. We have seen incidentally the result of Bockhart's experiment on the urethra of a dying paralytic, and the pathological investigation of the condition of the mucous membrane. Bockhart's conclusions have been warmly contested, especially with regard to the extraordinary penetrative power of the gonococcus, and its capacity for migration. But from the nature of the case there are few in a position to speak with confidence on the subject. Very few opportunities occur of submitting to sufficiently searching examination the urethral mucous membrane when it is in the condition to throw light on the pathology of gonorrhœa, and no opportunity at all of observing the progress of the disease in the urethra from day to day. The principal work of pathological investigation has been carried out upon the conjunctiva of the new-born; and here Bumm's[1] contribution stands out pre-eminent. Bumm had the opportunity of snipping out a small portion of the conjunctiva in a large number of cases of ophthalmia neonatorum at different stages of the disease. These portions of tissue were prepared and submitted to careful microscopic examination; and the observations thus made cannot fail to throw much light upon the minute anatomy of the disease-process in the genital tract. His observations extend from the first to the thirty-second day of the disease, when the gonococci could no longer be found either in epithelium or in connective tissue. The knowledge derived from a short summary of Bumm's conclusions may be advantageously applied analogically to the phenomena of the disease as observed in the female sexual organs. It will be found to explain some clinical features otherwise obscure or inexplicable, and to suggest a reasonable explanation of some symptoms of whose causation we have at present no direct knowledge.

Along with the infecting secretion at the outset of the purulent ophthalmia infection, there comes in contact with the conjunctival sac a small number of the micro-organisms. These find in the superficial moist layer of the conjunctiva an excellent soil, and proceed at once to proliferate, so that in

[1] *Loc. cit.*

twenty-four hours from the first contact they may be found in great abundance. While other micro-organisms which may have been brought incidentally into the conjunctival sac, amidst the infecting secretion, are rapidly eliminated by the movements of the eyelids and the flow of tears, the gonococcus holds its ground. "Endowed with an eminent invasive force," it penetrates between the superficial epithelial cells, and also into the soft protoplasmic substance of the same elements, continues to proliferate between the layers of the epithelium, and ultimately reaches the papillary layer of the mucous membrane. There is no evidence that the micro-organisms possess any power of locomotion to carry them against the outward stream of fluid. They develop superficially and inwards, probably owing to something in the nature of the soil. The time taken by the process varies to some extent, but usually in two days the micro-organism has penetrated and taken complete possession of the whole epithelial sheath. The way in which it marches forwards is always the same—by breaking down the cementing substance between the epithelium elements. Sometimes the gonococci are seen pressing forward in thin streaks, sometimes spreading out into rounded colonies or thinly scattered groups of fungi, according to the resistance of the tissues to be penetrated. In every case the reaction on the part of the tissues is at this time very energetic and complete. Great swarms of white blood-corpuscles migrate from the capillary network which comes close down to the epithelial sheath, penetrate into the superficial layers of the connective tissue, and thence find their way through the epithelial layers to the surface. Owing to the disintegration of the strata of epithelium by the development of the fungi, it gets carried away in the stream of fluid and of cellular elements, either as individual epithelium cells or in flakes, a breaking up to which capillary hæmorrhages between epithelium and connective tissue and the movements of the eyelids contribute.

At the verge of the transition-epithelium of the limbus corneæ and of the edges of the eyelids, the invasion by the fungi breaks off suddenly. This epithelium, as well as the pavement epithelium of the cornea, retains its regular stratification and normal appearance. It resists the invasive force of the gonococcus.

When the epithelial sheath is broken through, the papillary portion of the mucous membrane lies exposed to the ravages of the micro-organisms. But their invasive force appears to be broken; they penetrate only the most superficial sheath of the sub-epithelial connective tissue. Usually the gonococci lie in small groups in the interspaces between the strains of fibres which run parallel to the surface. More rarely they may be seen in streaks penetrating perpendicularly, and these possibly reach the capillary lymph-spaces.

While the micro-organisms are thus invading the superficial layers of the conjunctival connective tissue, the inflammatory process appears to have increased in intensity; the infiltration of round cells reaches finally as much as 2 mm. under the free surface of the mucous membrane, and the individual cells are crowded upon one another. This process gives direct origin to the purulent stage of the blennorrhœa.

The process of repair begins usually within four days, but it varies according to the severity of the attack. It starts from the remnants of the original epithelial covering, proceeds rapidly, and soon brings to an end the further development of the cocci in the tissues. The new epithelial sheath consists of a layer of cubical or slightly elongated elements, which becomes rapidly covered with two or three layers of flatter cells. The most superficial of these layers becomes further modified, and the whole sheath forms an impenetrable protection to the underlying tissues against the micro-organism. By the time the process of repair is well advanced, viz., the tenth or twelfth day, the gonococci are no longer present in the connective tissue, only in the superficial portion of the epithelial sheath, where whole ranks and groups of the micro-organisms still hold their ground. Occasionally there is a re-invasion of the deeper strata, when the new protective layers are broken through at points by the outward stream of *débris* from the surface of the connective tissue, and then there is a fresh invasion by the cocci, a repetition of the process, and, to some extent, of the appearances resulting therefrom. Such relapses hinder the process of repair, but do not prevent the complete development of the new kind of epithelium. The process of epithelium formation is usually complete in the third week of an attack of ophthalmia of the new-born, and the epithelial sheath remains intact during the subsidence of swelling and proliferation of elements in the papillary layer behind it.

The force of the invasion by the fungi and the duration of its different phases vary greatly in different cases. When the processes in the ophthalmia of the new-born were compared with the stages of the gonorrhœal ophthalmia in the adult, it was found that the process of repair was longer in beginning in the latter, and the invasion of the connective tissue of the mucous membrane was deeper, more extensive, and of longer duration. As late as the fourteenth day of the disease in an adult no trace of an epithelial sheath could be seen; the papillary portion lay completely exposed, and contained, to a considerable amount, colonies of gonococci, partly free and partly in the cell protoplasm of the pus corpuscles.

When we see the invasion by gonococci giving rise to such different phenomena according to the age of the individual attacked, we cannot reject, without further consideration, the view that when implanted upon such a suitable soil as the urethral mucous membrane of the adult, the gonococcus may develop more vigorously and penetrate more deeply than it has been shown to do in the conjunctiva of the new-born. It is not improbable, though there exists no direct proof except in the single case of Bockhart, that the invasion may extend to the lymph-vessels and even into the capillary blood-vessels, but such an occurrence must be the exception, not the rule. Bumm maintains that he has proved that the gonococcus can only invade and destroy cylindrical epithelium, it cannot enter the pavement epithelium, and it can penetrate only the most superficial layers of the connective tissue of a mucous membrane. Whether there is any essential difference in the mode and extent of invasion when the gonococcus develops in the mucous membrane of the urethra, of the cervix uteri, or of the Fallopian tube, remains to be proved by direct observation. As far as our knowledge of the process in the conjunctiva is concerned, we see that the reaction of the tissues in the acute stage corresponds exactly to the violence of the onset and the rapidity with which the micro-organism is developed; and in the later stages, when the tissues have become protected by the development of epithelium due to the reaction of the tissues, and the gonococcus disappears, the remaining phenomena of the disease are just those which may be produced by any chronic irritation. The final process of healing depends not so much upon the elimination of the micro-organisms, as

upon the closing of the tissues against their further invasion, by the development of thick layers of pavement epithelium.

The application of these conclusions to the phenomena of the gonorrhœal process in the genital tract is so obvious that any attempt at an explicit statement would be superfluous. The reaction of the tissues leading to the development of a special modification of epithelium, which will ultimately become in some sense cicatricial, explains at once the modified symptoms of second and subsequent attacks of gonorrhœa; and there are other equally striking points in the fitness of the results of Bumm's observations to explain clinical features of the disease, as we must trace them in the sexual organs.

Theory of "Mixed Infection."

No account of the pathology of gonorrhœal infection would be complete up to the present time without mentioning in some detail Bumm's theory of mixed infection, which has been already hinted at. His most recent contribution is a paper[1] published in December last, which, we may take for granted, contains the most mature experience on the subject.

Mixed infection is defined as the invasion of the organism by two or more kinds of bacteria. When these mixed infections are accidental they deserve no further notice. But that kind of mixed infection in which there exists a causal connection between the invasion of the two germs is most important. In such a case the bacterium possessing the greater power of invasion precedes and, as it were, prepares the soil for the second germ. These forms of mixed infection are distinguished by a certain constancy of occurrence, and the possibility, and even probability, exists that if one germ has settled another will follow. Ordinary croupous pneumonia with its after diseases is a typical example. The bacteria of pneumonia deprive the alveoli of the lungs of their epithelium, and fill them with an exudation, which is a soil in which secondarily tubercle bacilli or pyogenous germs settle, and which may lead to the termination of the illness in phthisis or formation of abscesses. The gonococcus, too, by its growth on the mucous membrane of the female genital tract, is capable of so altering its substratum that other fungi which are usually present there

[1] "Ueber gonorrhoische Mischinfectionen beim Weibe."—*Deutsche medicinische Wochenschrift*, No. 49, 1887.

without doing harm, or which, favored by suppuration, find their way from the air, obtain an entrance into the tissues, and then on their own behalf exert their specific effects, which are totally different from those of the gonococcus.

This fact is well illustrated in the gonorrhœal infection of the vulvo-vaginal glands. These and their ducts are tolerably frequently the seat of gonorrhœa in women. The ordinary course is this: an acute purulent stage is followed by a chronic mucous discharge, which may continue for months. The secretion gradually ceases, part of the gland becomes destroyed, and finally cure supervenes. The course, however, is totally different if, during the course of the acute blennorhœa, pyogenous micro-organisms find their way into the inside of the gland. The previously only tolerably tender gland becomes painful and swells, the skin covering it becomes red, and soon the appearance of purulent Bartholinitis is produced.

Such a gland, excised and examined, shows no traces of gonococci. The pus inside the gland only contains pyogenous staphylococci; these have overpowered the gonorrhœa microbes, have penetrated into the gland-sac, and from there into the periadenitic connective tissue. This is covered thickly with heaps of cocci, and shows the first commencements of purulent liquefaction.

The great majority of all abscesses of the vulvo-vaginal glands are due to this kind of gonorrhœic mixed infection. Other fungi possessing no pyogenous properties may invade the gland without giving rise to any characteristic tissue reaction. Similar processes occur in the mucous follicles of the female urethra.

Catarrh of the bladder is another form of gonorrhœic mixed infection. Gonorrhœic cystitis does not exist in the sense that the specific cocci of gonorrhœa attack the epithelium of the bladder, as they do that of the urethra, and then cause it to suppurate. Bumm has isolated the active agent in cystitis—a special kind of coccus which is very similar to the ordinary gonococcus.

Returning to the genital tract, the inflammatory processes which, after cervical and uterine gonorrhœa, usually develop in the pelvic connective tissue and pelvic peritoneum are extremely frequent. Bumm says: "It is astonishing with

what frequency here (Würzburg), in gonorrhœa patients, in whom indeed excesses of all kinds, continuous mechanical insults, &c., but often also a too heroic (eingreifende) treatment tend to help, para and perimetritic processes are found." The microbes of gonorrhœa do not directly invade the lymph tracts and the connective tissue about the uterus, and cause inflammation and exudation. The territory of the gonococcus is fortunately limited to the superficial layers of the mucous membrane. Gonorrhœic para and perimetritis cannot be explained by the action of gonococci; there must therefore be another agent, which, under the influence of the infection of the mucous membrane, passes into the parametric tissues and there causes disturbance. It is not very common for that exudation to break down into pus; if it does so, the occurrence is due to mixed infection. Bumm has had two such cases: in one he demonstrated the staphylococcus pyogenes aureus; in the other, the noxious agent was a chain micrococcus. In both cases the formation of pus was doubtlessly due to the pyogenous micro-organisms, which found admission through the infected and eroded cervical mucous membrane, and settled and multiplied in the lymph spaces of the broad ligaments till they were eliminated by the purulent inflammation. Bumm thinks himself, therefore, justified in saying, "Purulent parametritis with gonorrhœa of the cervix is due to a mixed infection with pyogenous bacteria; it is the analogue of the acute gonorrhœic bubo in the male, which likewise owes its origin to pyogenous germs."

The further the gonorrhœal infection advances upwards from the cervix, the fewer admixtures occur in the gonorrhœic pus. In gonorrhœic uterine secretion, besides the gonococcus, but very few other bacteria are found, and they are scarcely ever found in the Fallopian tube. The gonococci advancing along the mucous membrane ultimately reach the tube, while other germs remain behind.

The proper character and the results of the pathogenous activity of the gonorrhœic microbes is therefore seen—pure and unadulterated—in the tube. They cause purulent inflammation of the mucous membrane, but the surrounding connective tissue remains free from them. The gonorrhœic tubal pus is evacuated into the peritoneum, and whereas in other conditions the bursting of an abscess into the abdominal cavity

is followed by the gravest consequences, in this case the whole process terminates with a circumscribed inflammation, encapsuling the exuded pus. The cause of this difference is the varying pathogenic value of the organisms which are contained in the pus. A puerperal pelvic cellulitic abscess bursting into the peritoneum causes general peritonitis, because it contains pyogenous streptococci, which rapidly multiply in serous cavities, and are capable of exerting the most deleterious effects. Gonorrhœal tubal pus cannot do this; its microbes do not find in the peritoneum conditions for their increase; the pus, therefore, acts as an aseptic body, becomes encapsuled, and is finally absorbed.

It may, under special circumstances, be possible for pyogenous cocci to get mixed with gonorrhœal pus, the result being a dangerous peritonitis. But such cases are as yet unknown or unreported.

Bumm then mentions another form of gonorrhœal mixed infection, viz., that with tubercle bacilli. Statistics, which are however few, and therefore not to be quite relied upon, seem to show that in the etiology of localised genital tuberculosis, gonorrhœic processes, especially epididymitis, play a *rôle*. The question arises whether the cases of isolated tubal tuberculosis are not due to an old tubal gonorrhœa. The thickened tubal pus, in which the gonococci gradually become disintegrated, might be a good soil for the tubercle bacilli, and so, by means of mixed infection, a tubal gonorrhœa might be changed into a tubal tuberculosis. He mentions the facts of a case which seem to support this opinion.

In the paper of which the above is a summary, Bumm frequently refers to parametritis as a result of gonorrhœal infection. It may be as well to take this opportunity of stating that hitherto my experience has not brought me a single case of parametritis in which there was the slightest evidence that gonorrhœal infection, pure or mixed, had anything to do with its production. While perimetritis is one of the commonest of complications, if indeed it is not part of the normal course of the disorder, parametritis must be among the rarest.

CHAPTER IV.

CLINICAL PHENOMENA OF GONORRHŒAL INFECTION IN WOMEN.

COMING now to the clinical phenomena of the disease, we shall see how far our modern doctrine of the pathology furnishes a feasible and sufficient explanation of the clinical signs and symptoms, and also to what extent our knowledge requires to be supplemented. It is not intended to reproduce here a description of an attack of gonorrhœa in the female such as might be suitable for a systematic treatise on the subject, but rather to describe and call attention to some features which are of value in relation to the diagnosis and treatment of the disease, but which, as a rule, do not attract sufficient attention. At this stage the description of subjective symptoms is purposely omitted to prevent repetition when we reach the illustrative cases which will be introduced.

There are two distinct types of the gonorrhœal disease in women: (1) the acute, and (2) the chronic or "creeping."

IN THE ACUTE FORM.

The acute form, as affecting the urethra, does not differ materially from the gonorrhœal urethritis of the male. There is the same reddening, swelling of the mucous membrane, itching and burning, and finally the same discharge and scalding in micturition. The troubles in the female are less, as a rule, than in the male, and the complications of the urethral form less serious. Hence the comparatively little notice the disease received while the urethral affection was believed to be the most important part of the series of phenomena which ordinarily result from infection. In most of the cases to which I shall have to draw attention, the urethral troubles passed away spontaneously, and at an early period in the

course of the disease. In the female the urethral form never occurs without other portions of the genital tract becoming involved, but the converse proposition is not true; the uterus may be affected, and the most serious complications may develop in the pelvis, without the patient ever having noticed any discomfort in micturition, and without the medical practitioner who may be called in to treat the complications being able to satisfy himself that any change in the mucous membrane of the urethral meatus has occurred. As a rule the vulva becomes next affected. The nymphæ become thickened and stiff, the mucous covering becomes eroded, and the exposure of the underlying tissues thus produced gives rise to much of the suffering in micturition. At the same time the orifices of the glands of Bartholini become inflamed, and similar tissue changes take place in those about the urethral orifice. In the ordinary Bartholinitis the disease spreads inwards and the glands become painful and swollen, but if the ducts do not become obstructed the swelling subsides, and a discharge from the mouths continues for some time in much the same way as from the urethra. Although the ostium vaginæ becomes red and inflamed in an acute attack it is never so deep red as the orifices of the ducts of Bartholini's glands. In every stage of the disease the peculiar reddening, sometimes with erosion, at and near these orifices, is always distinctly visible. The reason of this appears to be that the mucous membrane about the ostium vaginæ, especially that within the remnant of the hymen, does not readily take on a diseased action; it is not a suitable soil for the development of the gonococcus. In women who are fastidious as to personal cleanliness you can hardly ever see any marked signs of vulvitis. In the careless, on the contrary, you not only find vulvitis, which may be due to the development of the gonococcus, but you find an erythema of the external surfaces of the labia and of the perineum which cannot possibly depend in any way upon the specific micro-organism of gonorrhœa, except inasmuch as the increased discharge due to the presence of the micro-organism becomes acrid and irritating, even to the external integument when left in contact with it. It stands to reason, therefore, that much of the tissue change which occurs in many cases of vulvitis depends more upon filth than upon anything specific.

It is not unusual to hear the acute form of gonorrhœa

referred to as vaginitis, by a sort of euphemism, and yet it is still a question whether there is any such thing as gonorrhœal vaginitis. The vagina seems to be the last portion of the genital tract, from the uterus downwards, to become affected, and the first to get well under any suitable cleansing process. Why should this be? Any one who has seen the uterus when a state of prolapse has been for long its usual position, cannot have failed to be impressed with the similarity of the vagina to the external integument. It has no genuine mucous membrane, merely a comparatively slight modification of the outer skin, and its surface is covered, like the outer skin, with a more or less horny epithelium. Such a surface is not easily invaded by the disease-process, and, indeed, in even well-marked cases of urethral and uterine gonorrhœa, it is sometimes hardly possible to detect any changes in the vagina. Schwarz[1] maintains that a vaginal gonorrhœa is not rare in the acute form, and he says he has seen it in cases where the uterus remained intact, and the vaginal changes could not therefore be attributed to contact of irritating discharges. But the discharge whch produces the erythema may come from outside, from beneath. If a woman is suffering from an extensive urethral and vulvar inflammation, with œdema and erosions of the nymphæ, the discharge will be copious, and flow into the vagina when she is lying in certain positions, unless the ostium vaginæ be very firm and contracted, which is not its usual state in the class of case under consideration; and in this way the infection may be set up and the symptoms increased by the production of an erythema from irritation, such as has just been alluded to as affecting the skin near the genitals under certain favoring circumstances. The younger the female affected the softer is the vaginal lining, and the more readily does a true gonorrhœal invasion result; we may therefore consistently maintain that a true gonorrhœal vulvo-vaginitis of the adult is rarely or never seen, and yet accept the evidence with regard to the specific nature of the vulvo-vaginitis of young children. Schwarz says that gonococci are to be found in and under the deeper epithelial layers of the vaginal lining, but he does not express himself so as to imply that he has found them. If, however, this turns out to be a

[1] "Die gonorrhoische Infektion beim Weibe."—*Sammlung klinischer Vorträge*, No. 279.

true observation, corroborated by capable observers, it follows that a genuine gonorrhœal vaginitis does exist.

On the other hand, and the fact may be referred to only as showing the difference of opinion which perhaps still exists, Martineau[1] does not look upon vaginitis as a directly specific disease. He describes the appearance of the vagina as seen through the speculum—the intense redness, the prominence of the plicæ, the layer of greenish pus covering the mucous surface, the small erosions, "superficial, red, and readily bleeding, which result from the desquamation of the epithelium produced by maceration in the vaginal fluid," and then he goes on to say: "I consider the vaginal granulations as an anatomical expression, as a clinical modality of the inflammation of the mucous membrane of the vagina, and not as the expression of a lesion proper to a virulent affection, blennorrhagia, or to a physiological condition, pregnancy." The vaginal granulations, according to him, merely tell us that the patient is affected with a constitutional malady or diathesis, such as the scrofulous, arthritic, or herpetic. This statement does not seem to add much to our knowledge, but clearly implies that the author, who speaks of having made observations on over 2,000 cases of blennorrhagia in women, is not satisfied with the ordinary explanation of the phenomena of the disease as it appears to affect the vagina.

Continuing the consideration of the parts affected by the acute form of the disease, we now come to examine the question of uterine gonorrhœa. The existence of a gonorrhœa of the cervix was formerly denied. Then its existence began to be recognised as possible; now, all the most recent observation goes to prove that *it is the rule, not the exception.* For my own part, I have seldom failed to demonstrate its presence by the examination of the muco-purulent discharge taken direct from the os uteri in recent cases, which were proved to be gonorrhœal by independent evidence. In uterine gonorrhœa, the mucous surface of the vaginal portion undergoes changes very similar to those referred to as affecting the inner surfaces of the nymphæ. These are reddening, swelling, and punctiform erosions. But however red this surface of the uterus may become, we can always see that the lining of the cervix is still

[1] "Leçons cliniques sur la blennorrhagie chez la femme," p. 72.

a deeper color, and that it is swollen, perhaps bulging out from the os. The inflammation spreads upwards in the cervix, giving rise to a copious purulent or muco-purulent discharge; and that it spreads to the cavity of the uterus we may infer, in the absence of direct observation, from the enlargement which takes place, and from the change in the type of menstruation, which is a uniform, or almost uniform, consequence of uterine gonorrhœa. It is this copious discharge which so frequently produces those changes in the vaginal mucous membrane over which it flows, and even in parts more external, which have been referred to as questionable evidence of a true gonorrhœal affection of those portions of the genital tract.

In describing the topography of the area of infection in a typical case of acute gonorrhœa, we might think it necessary to stop with the invasion of the cavity of the uterus. But, whether typical or not, there can be no doubt that in a very large proportion of cases the processes of the disease extend still further.

The process extends to the Fallopian tubes, ovaries, and peritoneum. Why this extension should occur in some cases and not in all, I cannot suggest any explanation further than by calling to mind what is implied in the varying vitality of the infecting organisms, and in some idiosyncrasies in the individual attacked, the consequently variable effect that might be anticipated from the varying initial force of the onset, and the nature of the soil, favoring or otherwise the development of the gonococci. In any complete investigation of this point, it would also be necessary to eliminate, as far as possible, all cases in which the gonorrhœal infection had already occurred even in an almost imperceptible "creeping" form. Between the virgin adult and the harridan subject of *colica scortorum*, there must be every shade of susceptibility. It cannot be a question of the narrowed condition of the uterine canal preventing the free flow of the discharge away from the orifice of the Fallopian tubes, for we see some of the worst results of extension of the process in women who have been recently confined, or in whom there is some subinvolution of the uterus with wide canal. Whatever be the causes of variation in the extent of invasion, we find frequently that acute gonorrhœa spreads along one or both Fallopian tubes. Here the process must destroy the ciliated epithelium, and give rise to a collec-

tion of fluid, if the canal does not remain patulous. We do not know by direct observation what goes on in the tubes, but we know that the processes of tissue reaction against the invasion of the fungus give rise to hydro and pyosalpinx, and to the permanent closure of one or both ends of the tubes; and we may suppose that occasionally the changes are so slight as to permit of resolution, and the return to a comparatively normal condition. We see something similar in the glands of Bartholini, where the disease, in an almost closed cavity, gives rise to pain and swelling, ending usually in resolution with a mild chronic disease of the part, but sometimes in complete closure of the duct, and the formation of abscess.

Whether the process of gonorrhœal disease, having once reached the tubes, ever stops short there, it is hard to say. The probability is, that it always extends more or less to the peritoneum. We know that the salpingitis causes enlargement and lengthening of the tubes. It may, therefore, produce adhesions by peritonitis originating in the extension of the disease perpendicularly through the walls of the tubes, just as peri-urethral phlegmon may result from urethritis; but it is much more likely that the peritonitis results from the continuation of the process in the same way as it has thus far extended, or by the discharge of fluid from the end of the tube. If there be a special acute gonorrhœal form of peritonitis, it spreads, as a rule, very rapidly over the pelvic peritoneum. The pain produced by the extension to the peritoneum is sometimes the first thing that attracts attention to the nature of the disease. The process has extended so far painlessly, but almost suddenly an aching begins in the pelvis, rapidly increasing to severe pain, and on an examination being made, perhaps within twenty-four hours after the first apparent symptom, the whole pelvic floor is found to be hard and immovable. In cases in which the peritonitis is set up by a sudden discharge of a coccus-containing pus or mucus from the free end of the Fallopian tube, it is conceivable that death may rapidly ensue from the shock. Noeggerath describes a case of almost sudden death from shock, which he attributes to the extension of the gonorrhœal process, but it is highly probable, nevertheless, that his patient died from perforating ulcer of the stomach. How violent the onset of the stage of peritonitis may be without any symptoms of shock is illustrated in a hospital case

under my care in August, 1887. The temperature chart is before me, and at the time the patient had to take to bed, after complaining for two days, the temperature in the axilla was 103.4°, and in the vagina 105.2°, the constitutional symptoms being in proportion to the pyrexia.

When the disease extends to the peritoneum it immediately attacks the serous covering of the ovary, and extends some way within the capsule. It gives rise by the tissue reaction to very considerable enlargement of the ovary, and, as the process of inflammation dies out, to thickening of its covering, and to more or less extensive adhesions to the neighboring viscera. How ovarian abscess may occur at a very early stage it seems by no means difficult to explain. We know that the ovaries of adult women often present considerable cysts or cavities without producing any symptoms, and while continuing to perform their functions in a perfectly physiological manner. These are the changes in the ovaries which are so often pointed to as a justification *post factum* for their removal, when no better justification is, or can be, forthcoming. Now supposing one of these cysts, with limpid contents, is situated near the surface of the ovary when the gonorrhœal process begins, its walls or a portion of them must become involved, the fluid contents increase and become turbid, and the cyst must either burst or become an abscess, just like the closed Fallopian tube, or the closed gland of Bartholini. Some such severe complications or accidents are required to explain the extreme amount of pelvic disease, and the persistency of the inflammatory processes, in a considerable proportion of cases. While the process can be observed beginning, and, as far as touch is a criterion, ending, in a few weeks in the milder cases; in the severer sort very grave symptoms are set up, and the disturbance of function, along with indications of anatomical changes, continues for months and even for years.

As bearing upon this portion of the subject, and indicating to what extent these opinions have taken hold among some clinical observers who have given the subject close and prolonged attention, I cannot do better than quote the following passages from Martineau's work: "The tubes are ordinarily affected in blennorrhagia, say the authors. They also frequently give origin to pelvi-peritonitis. Thus think Mercier,

and Bernutz and Goupil. A. Tardieu reports the case of a young girl, affected with vaginitis, who died of peritonitis. At the autopsy he found a metritis of the mucous membrane of the uterus, with suppuration; the tubes were filled with pus.

"With regard to pelvi-peritonitis, its existence is admitted by authors since the publication of the work of Bernutz and Goupil. In the sixteen months which they have devoted in this hospital to the study of this affection, there have entered ninety-three patients affected with blennorrhagia, and twenty-eight of those, that is to say, nearly a third part, presented this complication. In the tabular statement of the cases, one sees that the pelvi-peritonitis only develops when the discharge is already of long standing. These authors have never seen it produced by sympathetic action; it has appeared to them, in every case, to have been the result of the propagation by continuity of the inflammation from the vagina to the mucous membrane of the cervix, from thence to the body of the uterus, and then onward to the tubes. The morbid condition of these has become the point of departure of the pelvi-peritonitis."

It is only right to add that Martineau holds the directly opposite view from that reached by Bernutz and Goupil. He proceeds to intimate to his readers how large is the amount of material on which he bases his conclusions, and adds: "You will find by a close examination of the material that primary uterine blenorrhagia is extremely rare. In about two thousand cases I have seen it only ten times at most. You will find, further, that ovaritis and salpingitis are so rare, that I have not been able to pick out a single case. As to pelvi-peritonitis, I have found it only twice."

When opinions are so divergent as these, one, at least, of the sides to the controversy must be in the wrong. All the results of recent observation go to discredit Martineau's conclusions.

A general gonorrhœal peritonitis must be a very rare occurrence. The bowels and omentum evidently become matted together, and form a barrier beyond which the process cannot extend. It is thus confined to the pelvic peritoneum. This well-established clinical fact would seem to point to the conclusion that the extension of the inflammation is, as a rule, carried on, not by a gush of dammed-up gonorrhœal fluid from the Fallopian tube, but by some slower process, which brings the tissue reaction deliberately into play, giving time

for the raising of a barrier against the further advance of the process.

The position we have now reached, then, with regard to the acute form of gonorrhœa in women is that the ordinary typical attack extends to the cavity of the uterus. This is a position which I confidently maintain, and to the further proof of which I hope to make some contribution. Whether in the ordinary typical case the process also involves to some extent the tubes, ovaries, and pelvic peritoneum, there is still room for doubt; but I firmly believe that such an extension of the disease is by no means unusual, though it ordinarily disappears without recognition, or is attributed, without any serious effort at diagnosis, to inflammation of the bowels, or to some other cause. This class of case has been described so far as acute, but it is by no means a type of disease that runs a rapid course like the acute gonorrhœa of the male. We sometimes find that the peritoneum has been reached in a remarkably short period from the time of the patient's first contact with the infecting discharge, but as a rule the process takes from two to three months while it still may be considered in the acute stage. The external symptoms, those of the urethral or vulvar affection, have perhaps long disappeared, while the disease is still spreading on in the acute form in the uterus, tubes, or peritoneum. It may be best now, in order to support this statement with regard to the time required, to illustrate the course of the disease by mentioning the facts of two cases, which might almost be said to be taken at random, but for the selection of a case in which menstruation was suspended owing to lactation, for the sake of comparison.

CASE 15.—A hospital patient seen for the first time on the 10th of June, 1887. Her only complaint was a sense of weakness, which she attributed to a very copious vaginal discharge. She was confined four months previously, she is suckling the child, and she has not menstruated since she became pregnant. Two months after her confinement she began to suffer from scalding and frequency of micturition, but not to an extent to cause much distress. Though she had passed through her confinement without any unfavorable incident, the urinary troubles were supposed to be due to after-effects of labor. On examination it was seen that there was a profuse yellow discharge staining the clothing and adhering to the pudendal

hair. There was no redness or swelling about the urethra or vulva, except an intense reddening of the orifices of the vulvo-vaginal glands. The vagina was somewhat reddened in parts, but it was not tender, for the most careful examination could be made at the first interview, and a Fergusson's speculum introduced without evoking any complaint. The discharge was found to come entirely from the os uteri. The uterus seemed somewhat tender to the touch, the mucous lining was reddened, and there was considerable erosion round the os. There was no marked enlargement of the uterus; a slight subinvolution would have accounted for the whole amount. No complication affecting the ovaries or peritoneum could be made out by examination, and the anamnesis suggested nothing of the sort. This case might have been passed over as one of slow involution, with, perhaps, some amount of endometritis from some unknown cause if the discharge had been slightly less, but for the striking appearance of the orifices of the vulvo-vaginal glands. This led to examination of the discharge, and the gonococcus was found again and again in the most characteristic form. The husband failed to respond to my request for an interview. On the 18th and 19th of June the patient complained of pain in the hypogastrium, and it continued to a slight extent until the 24th, when she was again examined. No changes could be made out in the pelvic floor or in the organs, but the pressure required for the bi-manual examination caused more pain than usual. When last seen on July 12th, after a month's treatment, the patient was considerably relieved of her discharge, but there was still a sense of discomfort in the pelvis, without any coarse change sufficient to enable me to make out thickening or enlargement. Lactation and the consequent absence of menstruation appears to have saved this patient from the usual perimetritis. In any case, such process of disease as was going on and produced the symptoms of the 18th of June, nearly three months from the onset, bore still the mark of an acute disease.

CASE 16.—The husband of a lady, whom I had attended years ago in a confinement, came to me in the spring of this year (1887), and explained, with much circumlocution, why he feared that his wife had become affected with gonorrhœa. He himself was now almost well, as he had been under treatment, and the contagion must have been conveyed at the beginning

of his attack. His wife was very reticent as to her symptoms, he said, and he could give no exact details. A few days afterwards she came to consult me, and she then appeared to be very ill. I learned the following facts:—She was last confined ten months before, and the child was weaned a month ago. Her symptoms have lasted already for over three weeks, and began with scalding in micturition, but not to a severe extent, and without unusual frequency. This discomfort had passed away altogether since an abdominal pain came on four days ago. After being on her feet for two hours or more she began to have pain in the hypogastrium, and it has continued. She has resolutely gone about and attended to her duties, but has felt very languid and weak since undergoing the fatigue which was followed by the hypogastric pain. On examination, the vulva and labia were found to be smeared with purulent discharge, but without reddening or tenderness to touch. There was no sign of active disease about the urethra, but the orifices of Bartholini's glands looked *sore*, deep red, with area of lighter color extending outwards on the pale vulvar mucous membrane, and moist, as if the epithelium were eroded. The vagina was slightly reddened, but not tender, and the duck-bill speculum was used in the course of examination without causing pain. The os was open, with a considerable laceration of the cervix with red raw surfaces on the right side, and there were extensive erosions of the vaginal portion. There was a copious flow of purulent discharge from the cervix, and this was found on examination to contain gonococci in abundance. High up to the left, behind the broad ligament, an ill-defined mass, or an area of stiffened tissues, could be felt, pressure upon which caused considerable pain. The ovaries could not be made out, and no further examination was attempted. Her pulse was 126: temp. 99° in axilla. When I next saw the patient, a week later, she was just ceasing to menstruate. The flow had amounted to flooding, and she had felt ill all the time. On examination it was now found that the pelvis was full of an exudation mass of very hard elastic consistency. The difference of resistance on pressure between the two sides implied that the diseased condition affected chiefly the left side, and I have no doubt the obscure thickening which I had felt at the last examination was the beginning of the process.

Under treatment for nearly a month the patient improved,

and the mass began to feel more elastic and less hard, but she had again a flooding menstrual period, followed by an increase in the pain and the constitutional disturbance. The third menstrual period was not accompanied by any relapse. When last seen, less than a month since, she had still a profuse discharge, but the perimetritic exudation could be only obscurely felt, and there was little discomfort.

This patient had acute symptoms for certainly over two months; even though the perimetritis came on comparatively early, the chronic stage could not be said to be reached within the first three months. It may be mentioned incidentally that if this patient had become affected while suckling, and she had come under my care, I think I would have vetoed the weaning in order to save the patient from the damaging effects of menstruation, and in this way we might have succeeded in aborting the perimetritis.

In the Chronic or Creeping Form.

Coming now to the consideration of the chronic and creeping form of gonorrhœal infection in women, let us take the facts of a typical case. A young woman, a year or less after marriage, finds that her health has undergone a serious change for the worse. Whereas, before marriage, she was as sound in health and as active as in childhood, she has now lost her buoyancy, her sense of physical well-being, and is distressed by unwonted pains and discharges. She has a persistent leucorrhœal discharge; she suffers from dysmenorrhœa more or less severe, and her menstrual periods, which were formerly painless and regular in occurrence, duration, and amount, are now painful, profuse, and variable. We elicit a history of gradual change in the type of menstruation, and the slow invasion of a sense of distress of an indefinite kind, calling attention to the pelvic and abdominal regions; or there may be the history of an abortion followed by more or less severe inflammation; or the patient may have borne a child at full time, and after some form of puerperal illness, she may have experienced a persistence of pelvic troubles, with subsequent sterility. In any such case a physical examination will probably reveal certain pathological changes in the sexual organs. There is the reddening, already referred to, at the

orifices of Bartholini's glands; the uterus is somewhat enlarged to the touch, and pressure upon it causes pain, whether from its own condition or from the pressure being conducted to sensitive neighboring organs; the ovaries are enlarged, tender, and dislocated, and there is probably also a thickening and fullness about the broad ligament, implying a greater or less degree of disease of the tubes; and thoughout the whole pelvic floor there is conveyed to the muscular sense of the examining finger the impression of a deviation from health of the tissues; there is loss of elasticity in the boundary walls of the fornices, which is never felt unless the parts have been at some former time changed by an inflammatory process. Examination with the speculum shows that there is a discharge from the os uteri more profuse than normal, though it is not necessarily changed in appearance or in other characteristics; and there is probably some reddening and hypertrophy of the mucous membrane where it appears at the os.

Such are the chief points in a fairly typical case. But the symptoms and signs of the disease which I am attempting to describe vary greatly, from mere vague discomforts and slight derangement of the menstrual type to such a serious state of matters as is produced by pyosalpinx and its necessary concomitants. The symptoms and the apparent injury to health vary with the temperament and the social circumstances of the patient. If she can rest and be well attended to, there is only the inability to enjoy the more active occupations and amusements to complain of, and the injury to the general health as shown by the appearance and condition is comparatively slight. When the patient must exert herself to any considerable extent, and cannot rest even at menstrual periods, the effects of the disease are soon made evident. The superadding of nervous symptoms in long-standing cases, with perhaps no apparent relation to the original local troubles, is one of the common-place phenomena in gynæcology, and need only be mentioned.

The state of health just described is characteristic of gonorrhœal infection, and it is reached either through a more or less acute attack subsiding into the chronic form, or by the gradual development of the creeping form of gonorrhœal invasion, in which an acute stage either does not exist or altogether evades observation. How can we explain such

serious changes in the pelvic organs as we frequently find without any preceding acute attack of gonorrhœa? The answer brings us back to Noeggerath's latent gonorrhœa in the male. The virulence of the gonorrhœal infection appears to depend upon the number and the vitality of the gonococci contained in the infecting matter. In an acute attack, the number of gonococci in the secretion is at its highest, and it would seem that the micro-organisms developing in fresh and suitable soil, are in the most vigorous state of vitality; hence the infection is conveyed quickly and with certainty, and, if to a virgin soil, with the result of producing an attack of the same virulent type. At the other end of the scale you have the sort of attack produced by the infecting matter from a man who has been the subject of an acute attack many months, perhaps years, before. The gonococci are few and decrepid, probably altogether absent from the periodic emissions of a continent man. It is only the post-nuptial sexual excess that rouses them into sufficient vigor to be harmful. Even then the gonococcus, developing under a fuller blood supply and a changed innervation, but in a comparatively worn-out soil, has not sufficient vitality to gain a footing in any part of the female genital tract except the most favorable to its growth; and its *locus electus* is the cervix uteri. It would seem that it is from this point that the disease in a woman who has become affected through the "latent" gonorrhœa of her husband, almost invariably spreads upwards and downwards. The discharge produced by the tissue-reaction set up by the feebly active development of the micro-organism may carry the infection downwards, and cause some slight vulvitis, with itching or discomfort, and it is possible that in this way the urethra and the glands of Bartholini may become affected when the *materies morbi* is of medium vitality; but, as a rule, the disease spreads upwards only, and the first symptoms are those of an endometritis.

The further course of the disease is, as a rule, intermittent. Just as in the male the blood supply and innervation of repeated sexual intercourse may be required for the sufficient development of the gonococci to make them capable of fresh growth in new soil; so in the woman, the periodic blood supply of menstruation, and the *status menstruatis*, may be necessary for the further development of the disease, even when it

has taken hold. We find clincally that there are exacerbations at the menstrual periods, and we can, under favorable circumstances, trace the course of the disease as it attacks one part of the genital tract after another, each new departure appearing to be from a menstrual period. If the woman has become pregnant before the infection takes place she may go on to full time, and then it is only in the puerpery that the disease begins to spread beyond the cervix, giving rise to the gonorrhœal form of puerperal fever, and all its after consequences. If the pregnancy and the gonorrhœal infection occur in quick succession, it would seem that then the development of the micro-organism may become the cause of abortion; to the abortion succeeding the further spread of the disease, in whose course, in fact, the abortion is a mere episode.

When the gonococci have once invaded the glands of the cervix uteri, they appear to linger there as long or longer than they do about the deeper parts of the male urethra. Developing in a new soil, they are endowed with greater vitality than the comparatively starved stock from which they sprung, and therefore the woman who has become affected with even the creeping form of gonorrhœa is probably in a much more contagious state than the man from whom she contracted the disease. A very intricate case bearing upon this point will be mentioned directly.

This long-continued vitality of the gonococcus in the cervix uteri, as a cause of the spread of contagion, has been already incidentally referred to. It is a fact not sufficiently recognized by the framers of rules for the inspection of *puellœ publicœ* in some countries, and hence the whole proceeding is a delusion and a snare.

The critical reader will have observed that in speaking of the "latent" gonorrhœa of the male as the cause of the creeping form of gonorrhœa in the female, no mention has been made of the presence or absence of a urethral discharge in the infecting individual. Here there is certainly a hiatus in our knowledge. Several men whom I have had occasion to question have admitted to observing after marriage the occasional return of a discharge or a moistening of the meatus which had long disappeared; others had failed to observe anything of the sort. Careful examination by the surgeon may sometimes lead to the discovery of a pathological condition, even

when the patient quite honestly denies the existence of manifestations of it. Noeggerath, in the recent paper already quoted from (p. 22), says he has sometimes succeeded in a suspected case in bringing to light a drop of pus from the posterior portion of the urethra long after the patient believed all signs or symptoms of an attack to have disappeared. But, is a visible external discharge a necessary accompaniment of the contagious condition in the male? Probably not, and for the following reasons. The men whose wives seem never to escape are those who marry a few months after being "cured" of a gonorrhœa which has run a protracted course. These are the men who get nearly cured by several surgeons in succession; or who, after undergoing the routine medical treatment for a few weeks or months, without exercising the self-control essential to rapid and complete cure, let the affection take its course, and being no longer in pain, are satisfied if they can conceal the evidences of the disorder. Such men almost invariably become the subjects of posterior urethritis; and it is reasonable to suppose that long after the signs of active disease, including discharge, have disappeared from the anterior part of the urethra, the micro-organisms, hybernating as it were, in the posterior part of the urethra, may be roused to a point of vitality sufficient to enable them to be carried away, and to develop in a new soil, without giving rise to such local irritation in the anterior portion of the urethra as to attract attention. If we could get at all the facts of one of the mildest of cases, we should probably learn that there was some change in sensation, some slight deviation from the standard of perfect physiological action, which would point to the posterior portion of the urethra as the seat of a pathological process. The most careful examination might not lead to the detection of the gonococcus. Neisser says he has discovered it as long as three years after the acute attack, but most investigators have failed to find it a comparatively short time after the acute symptoms have disappeared. In this respect the majority of observations agree with the results of Bumm's examination of the various stages of the disease in the conjunctiva, where it was found that the gonococcus disappeared from the tissues at a certain stage of the process. The question then arises, In what form does it persist? And here again we reach the limits of our knowledge. Sänger has

suggested a spore, which has not yet been isolated, as the permanent form of the contagium in the tissues, but this is so far a mere hypothesis. The only fact which I can suggest in its favor is something which, so far, I have seen no reference to, and which may be a mere mistake in observation. In examining the discharge in a case of acute gonorrhœa with the highest powers, one is struck, not only with the vast number of diplococci, both connected with pus or epithelium and lying free, which are to be seen in the field, but also with the innumerable minute objects, call them granules or spores or *débris*, which are everywhere mixed up with the cocci. So far I have only seen this phenomenon in acute cases, and for aught that any one can as yet allege to the contrary they may be the spores of a fungus, such as we must see in tubercle were the bacilli to disintegrate and cast the spores about; and some portion of them may be destined to remain in the tissues, when the full-grown organism is cast out, until a fresh pabulum has developed or until there arrives a new stimulus to further growth.

I have repeatedly alleged that it is the neglected cases of gonorrhœa in the male—those which become chronic—which most frequently gives rise to the infection of the female, even though they may have long ceased to show signs of activity. If this statement can be proved, the fact has obviously very important bearings.

Proceeding on the plan hitherto adopted, I shall give here two specimen cases, which, of course, illustrate more points than the one at present in question.

CASE 17.—Hospital patient, wife of a working man whose occupation appears to be incompatible with personal cleanliness. Her married life, except a few months at first, is a history of constant bad health. The patient is now 38 years of age, and has been married six years. She had a still-born child at the end of the first year after marriage. She was attended by a midwife, and is said to have had a "low fever" after her confinement, and was ill for two months. Never been pregnant since. She had flooding and irregular menstruation for two years after her confinement, and one of these periods was supposed to be a miscarriage, but the assertion is founded on a mere midwife's diagnosis without the patient's knowledge of facts to warrant it. Menstruation is now

scanty; period usually one day. Great pain in hypogastrium begins at periods, and is said to be worse about a week after. Profuse leucorrhœa in intervals. On examination, the vulva was found to be normal; the uterus was apparently in the normal position, and very tender to the touch, but more careful examination later showed a lateral flexion; the left ovary enlarged and sensitive, right not made out. The speculum showed the cervix slightly lacerated and flattened, with erosion round the os; a thin yellow discharge, rather profuse. These facts were elicited at the first interview twelve months ago, and on a subsequent occasion it was discovered that there was narrowing of the internal os with a chronic endometritis and sensitiveness of the uterine canal. The patient has been under treatment ever since without any appreciable improvement. She is always ailing, but not so ill as, in my judgment, to justify me in proposing removal of the diseased ovaries and tubes, especially as I infer from the thickening of tissues in the pelvic floor and the deformity of the uterus that there must be strong adhesions of long standing. Suspecting the origin of the patient's troubles, I obtained an interview with the husband. I learned from him that he contracted gonorrhœa rather more than a year before his marriage, but he was not sure about the time. The pain was not very severe, and the discharge did not trouble him, so he "took no notice of it." It was better and worse for a long time. He took his "glass of beer of a Saturday night," but would not admit to any kind of excess. He rather thought he had some of the discharge shortly before his marriage, and he may have seen it sometimes after, but he was not certain. From the wife I could obtain no evidence of any symptoms that might not have been equally due to pregnancy.

My interpretation of the facts is that the wife became affected by a low creeping form of gonorrhœa after she became pregnant, and that the disease developed rapidly into a comparatively virulent form after parturition, with the results described.

CASE 18.—A lady, æt. 30, who, at the time she first came under my observation, had been married two months. Ten days after marriage she was seized with a sharp pain in the hypogastrium, and though she got up and dressed she could not walk, and had to spend some days on a couch. She and

her husband returned home, owing to her illness, and she then for the first time sought for medical assistance. The pain in the pelvis continued, and she felt so ill that she had to take to bed. She says she was treated for "inflammation of the bowels," and was under treatment four weeks, when she was considered cured. She had hardly attempted to attend to her domestic duties when the illness seems to have relapsed, but she went on for nearly a month without further treatment. It was then that I saw her for the first time. She looked very ill, and told me in answer to inquiries that she had lost flesh rapidly since the beginning of her illness. She still complained of a pain in the hypogastrium, in the groin, and round the loins. There was a profuse discharge, she said; but she used to have "the whites" before marriage, only the discharge was now much more profuse, and it was different in appearance. Since her marriage she has menstruated twice, both times very profusely, and for a longer time than formerly. On examination I found the uterus was fixed, with exudation which was most marked on the right side, but extended round behind the uterus and occupied the left half of the pelvis, where the inflammation seemed to have been less intense. It was not very tender on pressure. There was a profuse yellow discharge which bathed the external parts, but did not appear to have set up any intense inflammation about the vulva. I obtained some of the discharge for examination, and afterwards found the diplococcus in the characteristic relations, but the microscopic appearances were never those of a gonorrhœa in the acute stage.

I had an interview with the husband, and obtained from him a rather remarkable history. Seven years ago he became affected with gonorrhœa for the first time, and appeared to have had a very bad attack. He was in the hands of a quack and of several medical men in succession, and appeared to have got cured about twelve months after the first symptoms of the disease. He had been circumcised in his early youth or infancy, and there remained two sinuses in the foreskin, one of which ran parallel to the urethra, at the side of the frenum, and the other was rather more transversely placed. The former was about an inch deep, and the latter was much more shallow. Ever since the attack of gonorrhœa these sinuses had remained moist, and were sometimes irritable; he could

then squeeze out a little moisture, or a tiny drop of turbid fluid from them. Soon after his gonorrhœa was cured this man married. In the course of the first year his wife had a child, and died of puerperal fever. The child survived, and was under medical treatment from its birth; but as he himself had to be abroad on business, and, on learning about the death of his wife, remained away for several years, he could not give details concerning this period.

He never saw any sign of urethral gleet since his final cure before his first marriage, but about twelve months ago, owing to the irritation caused by the deeper sinus, he consulted a surgeon, who slit open and cauterised the canal. The smaller one was let alone.

Both husband and wife were perfectly well to all appearance at the time of the marriage, two months ago, and the illness from which the wife is suffering came on without any apparent cause. But since her illness the husband has begun to suffer from urethritis. It came on four weeks after marriage, and at the time of my first interview with him it had developed, though under treatment, into a rather acute urethritis, and it ran the course of a moderately severe attack of gonorrhœa. As he had been under treatment for three weeks or more, I was not surprised at being unable to detect gonococci in the discharge, and I entirely failed to discover any suspicious micro-organism in the minute speck of fluid which he could press from the orifice of the small sinus.

The question arises then, Whence came the infection to the woman? Of course, some accidental contact with a gonorrhœal poison is a possible explanation, but it is a marvellous coincidence that a woman of thirty should have escaped so long only to become accidentally infected at a time when the infection would be conveyed to her husband. From my knowledge of the facts, I would say that the suspicion that this respectable woman of thirty contracted a disease from another source just on the eve of her marriage may be peremptorily set aside. Besides, the infection did not act like ordinary acute gonorrhœa. In the woman the perimetritis came on as soon as the discharge, and the discharge did not readily infect the husband, unless the husband is simply lying. We seem almost driven to the conclusion, therefore, that the gonorrhœal virus lingered in the sinus for years, only to

develop actively when it reached the favorable soil of the uterine canal, and that it spread in the manner characteristic of "latent" gonorrhœa, but with exceptional rapidity.

The wife, who was kept under constant treatment for over three months more, considered herself well at the end of that time, but though the uterus was then movable, the thickening of the peritoneum was distinct. There was the peculiar loss of elasticity all across the pelvic floor, and the deep-lying sense of resistance. Probably both tubes were ruined, the fimbriæ matted together, or adherent to the ovary, and the abdominal orifices closed. There may be now no further complication; a sort of resolution has apparently gone on, and the patient may escape any of the more serious injuries to her health. But menstruation, which continued to be still too profuse, will never again be perfectly normal, and the closing of the Fallopian tubes can never be undone.

CHAPTER V.

CONSEQUENCES OF GONORRHŒAL INFECTION TO THE INDIVIDUAL.

In discussing the consequences which may, and most frequently do, arise from gonorrhœal infection in women, I shall not attempt to place the pathological changes in one series of paragraphs and the immediate and remote effects of these changes on the individual in another series, but mention them together, and that as concisely as possible. This method of treating the subject seems to possess some advantages, although to a certain extent it involves repetition.

Urethral and Vulvar Gonorrhœa.

The urethral form of the disease is exactly analogous to the same disease in the male, and lays the subject of it open to nearly all the same complications. From gonorrhœa in the female may result cystitis, disease of the ureters, and of the kidneys, just as is the by no means rare consequence in the male.

Chronic inflammation of the glands of Bartholini is almost too trivial an ailment for mention in the present relationship. Acute inflammation may be a very painful ailment, and when the disease has spread onwards this painful affection may be a somewhat serious hindrance to effective treatment. In a case which came under my care in October, 1887, the right gland developed so rapidly into an abscess that it was impossible to carry out the proper treatment for preventing uterine blennorrhœa until the Bartholinitis was thoroughly dealt with. This required chloroform narcosis and the use of the cautery, and then measures could be taken which prevented the further spread of the disorder. In only one case (which has been mentioned) have I been able to convince myself that

Chronic Bartholinitis was more than an inconvenience—viz., that in which a pessary, when it slipped a little, seemed so to press upon the tender spots corresponding to the site of the glands, as to add very considerably to the patient's sufferings.

Uterine Gonorrhœa.

The invasion of the cervical canal is marked by the appearance of a profuse mattery discharge. It would appear that a rapid change takes place in the epithelium both of the cervix and of the cavity of the uterus. The cylindrical epithelium is destroyed for the time being, and is probably never perfectly restored. Its place is taken, to some extent, by layers of a sort of pavement epithelium, in fact, a kind of cicatricial tissue. There thus occurs a deeper change than that which takes place at the menstrual period, profoundly modifying the fresh developments of the mucous membrane for the future. Without going into detail, these are the results obtained by Zeller[1] in his investigation into the subject of endometritis, and they are just the changes which we might expect from what we learn from Bumm's inquiry into the action of the gonococcus in the conjunctiva. The results obtained by Mr. J. Bland Sutton, in a recent investigation into menstruation in the human female and in monkeys, bring home to us more clearly the cause of a most important sign of gonorrhœal invasion of the body of the uterus. His conclusions are: 1. The uterine mucous membrane is normally not shed during menstruation, but only the epithelium. 2. The sanguineous discharge is due partly to oozing from the surface denuded of epithelium, and in part to active congestion. 3. The discharge from the uterus is largely augmented by mucus secreted in increased quantity at this period from the enlarged utricular glands.[2] Keeping this in mind as the physiological process, we can well understand that the superadding of the tissue reaction produced by the gonococcus would at first merely exaggerate the amount of menstrual flow, whatever the remoter effects might be. The pain and nervous derangements which ultimately super-

[1] "Plattenepithel im uterus (Psoriasis uterina)."—*Zeitschrift für Geburtshilfe und Gynäkologie*, Bd. IX., Hft. 1. Summary in *Centralblatt für Gynäkologie*, No. 10, 1885.

[2] *British Gynæcological Journal*, Part VIII., Nov., 1886.

vene are probably due to changes in the tubes and ovaries as well. To the later changes in the lining of the uterus, when it assumes its almost cicatricial character, are probably also due the remoter changes in the menstrual discharge, which, instead of being profuse as at first, becomes scanty in amount and sometimes changed in character. In addition to the endometritis there comes on a greater or less amount of hyperplasia of the uterus, and with this there must necessarily be some derangement of innervation. The sense of dragging and sacralgia in the early stages is not probably from mere weight alone.

The increased menstrual flow is such a constant feature in uterine gonorrhœa, that I shall introduce here two typical cases. In one of them it was the sudden change in menstruation, without apparent cause, in an unmarried woman, which excited suspicion and led to a correct diagnosis.

CASE 19.—P. B., æt. 23, single, domestic servant, residing in Preston, sent to me by her mistress on November 9th, 1887. She was suffering from pain in the hypogastrium, supposed to be the result of a kick from a refractory child. There was a history of good health up to six weeks since, when her symptoms began. She had a rather sudden attack of severe pelvic pain four weeks ago. Her last menstruation was unusually profuse and continued for a fortnight. Since then there have been mattery discharge, hypogastric pain, etc. My diagnosis, after examination, was gonorrhœal endometritis, salpingitis, ovaritis, and perimetritis of the left side; and I elicited information from the patient which proved the diagnosis correct. She felt very ill, and I advised her to seek admission into Preston Infirmary, under the care of my friend Dr. Garner, to whom I wrote. She was accordingly admitted on Nov. 17th. Dr. Garner has kindly sent me the notes of the case, from which I select the points relevant to my present purpose. The patient was under treatment till the 13th of January, when she was dismissed cured. She was kept rigidly to the recumbent posture, and suitable measures were taken to diminish the congestion of the pelvic organs. Three days after admission the vaginal temperature became normal. She began to menstruate on Nov. 28th, and continued till Dec. 7th, extract of hydrastis having been administered from the 5th, owing to the excessive hæmorrhage. The general and local treatment

was continued until the 1st of January, when she began to menstruate again with much less pain. The period lasted only till the 5th. Examination on the 11th of January showed an entire absence of pain on pressure, though there was still considerable thickening of the tissues in the left half of the pelvis. The uterus, which had been fixed, had begun to recover its mobility.

CASE 20.—R. N., æt. 21, single, living at home with her parents, who are of the respectable working class, applied for treatment at the Manchester Southern Hospital, on February 10th, this year (1888). After hearing several irrelevant and misleading statements as to the patient's past and present state of health, and the treatment she had undergone, I learned that she had had a profuse and prolonged menstrual period, without any apparent cause, at or about Christmas. This fact led to some pointed inquiries and a thorough examination, about which the condition of the pudenda indicated there need be no scruples, the result being the establishment of the diagnosis of perimetritis with salpingitis of both sides. The patient, after admitting certain facts, answered questions frankly enough, and finally the following points were settled: —(1) Scalding in micturition began four months ago; (2) the first profuse menstruation occurred at Christmas, and the last period, which ended a week before the date of our interview, was also extremely profuse; (3) the hypogastric pain began at the time of the profuse period at Christmas; (4) she felt so ill again, just after the last period, that she had to take to bed, and she had got up, the first time for a week, to come to the hospital. The cause of the increased pain and feeling of illness was no doubt a menstrual exacerbation in the perimetritis. But the sudden, apparently causeless, increase in the menstrual flow is the chief feature of the case, as an illustration of the point under consideration.

TUBAL GONORRHŒA.

But by far the most important changes in the internal sexual organs which result from the gonorrhœal infection are those affecting the Fallopian tubes and ovaries. With regard to the tubes, it would seem that the effects vary greatly, according to the virulence of the infection. If the virus is in a state

of high vitality, the disease may spread so rapidly along the tubes that the abdominal ends become sealed by the peritonitis before the active stage of tissue reaction has ceased in the course of the tubes. The consequence is that the tube becomes distended by the collection of fluid that forms, thickened in its walls, and perhaps lengthened as well. We thus reach the condition of hydrosalpinx, which will, under favoring circumstances, sooner or later, advance to the still more serious condition of pyosalpinx. When the process of invasion of the infection is slow, as in the creeping form, we very seldom see it resulting in pyosalpinx. Yet there can be little doubt that many of the cases of puerperal pyosalpinx are the result of the assumption of a comparatively virulent type under favorable circumstances by a chronic or creeping gonorrhœa. Pyosalpinx is certainly not a rare consequence of gonorrhœal infection, and it does more serious injury to health and is more dangerous to life than any of the other complications. Yet the risk which the affected run of having their health ruined and their lives endangered by this condition is by no means even yet generally recognized and acted on by the medical profession in this country. As far as I can learn from the literature of the subject, Mr. Lawson Tait is the only English surgeon who has continued for years to call attention to this complication. He has taken occasion to refer to gonorrhœa, again and again, as the cause of the severer forms of Fallopian disease in cases where he found operation necessary, and even in his work on "Diseases of Women," published in 1877, frequent reference is made to gonorrhœal infection as a factor in the production of some of the more serious complications. He says,[1] in reference to ovaritis, "Acute ovaritis from gonorrhœa is a common result of the infection, and is a frequent cause of sterility. . . This affection, as far as is known, generally results in disorganisation of the gland and the formation of adhesions round it." He also recognises gonorrhœal infection as one of the most ordinary causes of perimetritis. In the edition of his work on diseases of the ovaries, published in 1883, he says:—"I have already said enough about hydrosalpinx and pyosalpinx to render it quite unnecessary to discuss them further than to insert here a complete list of all the cases upon which I have operated for these diseases. Both conditions are far more common than was believed previous

to my experience, yet they have been quite well known, and described for at least half a century. Of the forty-four cases only four have occurred in single women, and the leading feature in the history of many of the cases was an attack of gonorrhœa. In one case I had to operate in the acute stage of the disease, which had arisen, on the admission of the husband, from this cause."[1]

At a meeting of the Obstetrical Society of London, held on the 4th of May last, when Dr. A. H. N. Lewers read a paper founded on observations made on the pelvic organs, in a series of 100 cases, in the post-mortem room of the London Hospital, the essayist reported that disease of the Fallopian tubes—that is, pyosalpinx, hæmatosalpinx and hydrosalpinx—was found in seventeen cases, and he read notes descriptive of these. Dr. Galabin, in the course of the discussion on the paper, mentioned that at Guy's Hospital the pathologists had found in 302 necropsies of women only twelve cases of distension of tubes, and two of these were very trivial, amounting in all to a proportion of 4 per cent. The cases of chronic inflammatory disease about the tubes amounted to 9 per cent. Mr. Lawson Tait spoke in eulogy of Dr. Lewers's paper. He found the conclusions drawn from the post-mortem room, as regards causation, progress, prognosis, and treatment, identical with those which he had been preaching for about ten years on the basis of clinical experience. He confessed that it was somewhat a staggering thing to find 17 per cent. of the women who died in the London Hospital suffering from tubal disease, and this did not include those cases which suffered the most, in which there were adhesions between the ovaries and tubes to the surrounding viscera, more particularly the peritoneal layer lining Douglas's pouch, resulting ultimately in complete retroversion of the uterus with its appendages, and forming one of the most dreadful conditions which the gynæcologist had to deal with. When removed it was difficult for an unskilled pathologist to see anything the matter with them. Dr. Lewers had not included such cases, and they must have been numerous. *He thought the explanation of the higher percentage at the London Hospital, and the small group*

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[1] "The Pathology and Treatment of Diseases of the Ovaries." 4th Edition, 1883.

at Guy's, must be due to locality, and that gonorrhœa was more common amongst the poor at the East End than on the south side of the river.[1] I do not know if the speaker gave reasons for this final remark, but, to those who have no local knowledge, it may be as well to explain that the London Hospital contains, in the area from which it chiefly draws its patients, the localities where sailors most do congregate, and among the women who apply for its relief are the victims of the neglected venereal diseases brought into a focus from every sea-board slum in the world. The same state of matters prevails in such places as Liverpool and New York, and we have perhaps in this fact alone some explanation of the frequent occurrence of the more serious forms of tubal and ovarian disease in these cities.

In Germany, the man who has, perhaps, most persistently called attention to the more serious clinical features of the gonorrhœal infection in women, is Sänger, of Leipsic. His views may be concisely and conveniently indicated by a quotation from a recent paper read before the Chicago Gynæcological Society.[2] He says: "Salpingitis gonorrhoica is the only specific infectious form of salpingitis which is recognised as such by Lawson Tait, although he stops short of admitting that the gonococcus is the exciting agent. Without doubt the gonorrhœal is the form most frequently met with. This fact was clinically established as early as 1872, by Noeggerath, long before Neisser had discovered his gonococci, or Lawson Tait had performed his first operation 'for suppuration of the uterine appendages.' In Germany, I myself was one of the first gynæcologists who at our meetings showed the frequency of gonorrhœal salpingitis, emphasised its causal connection with pelvic peritonitis, and removed by operation the gravely implicated uterine adnexa. Gonorrhœal salpingitis is never followed by a 'destructive suppuration' of the uterine appendages; it remains invariably a disease of the surfaces of the mucous and serous membranes. The pus formed by the specifically diseased mucous membrane gradually distends

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[1] "Summary in the *British Gynæcological Journal*, Part X. (August, 1887), p. 273.

[2] "Salpingitis gonorrhoica et syphilitica."—*The Obstetric Gazette*, February, 1887.

the tube; in one class of cases, in which there is a great accumulation of free pus, the tube is transformed into a large sac with thin walls; in another, in which the wall of the tube, especially its muscular tissue, is hypertrophied to a greater extent, the tube becomes much thickened and rigid. In most cases both conditions are found, the uterine portion of the tube is thickened, the abdominal end dilated. The serous surface of the tubes, the albuginea of the ovaries, the serosa of the peritoneum are attacked or become pus-secreting surfaces only in cases in which gonorrhœal pus has escaped from the tubes and thus infected the above-named structures. . . . I do not believe that gonorrhœal pus ever penetrates the walls of the tubes and thus produces these diseases. But a specific gonorrhœal inflammation of the mucous membrane of the tube, with secretion of pus into the cavity of the latter, is accompanied by a non-specific inflammation of the entire tubal wall."

My present object is only to call attention to the most advanced opinion at home and abroad on the spread of gonorrhœal infection to the Fallopian tubes, and the consequences which result therefrom; it is not to assist in attracting attention to this polemic, much less to seek to take part in it. But if the illustrative case of pyosalpinx, which I am about to mention, does not also amount to a case of "destructive suppuration of the uterine appendages," it would be hard to describe the state of matters in English.

CASE 21.—Mrs. B., the wife of a well-to-do operative in a Lancashire town, is 34 years of age; has been married seven years, and has never been pregnant. She has always suffered from dysmenorrhœa since puberty, and this continued unchanged after marriage. Her husband contracted gonorrhœa six months ago, and at once infected his wife. Both husband and wife were under the care of the same practitioner, their family doctor, at the same time, the wife being ignorant of the nature of her ailment. She had a very severe acute attack, and about six weeks after the first symptoms, she completely broke down at a menstrual period, and had to take to bed. She was in a state of fever, and complained of intense pain in the pelvis, chiefly affecting the left side, and extending upwards towards the crest of the ilium, and down the thigh. The doctor found the uterus fixed, and the pelvic floor hard, rigid, and still painful. She was under treatment at home for four

months, during which time she was chiefly confined to bed; she lost flesh, had night sweats to slight extent, and suffered almost constant severe pain in the pelvis, with nocturnal exacerbations. When she first came under my care, five months from the time of the infection, she was still in the condition just described. The uterus did not seem much enlarged, and the exudation appeared to be most marked on the left side of the pelvis. The chief pain was still in the left half of the pelvis. The profuse yellow discharge, which was said to have existed at one stage of the disorder, had almost completely disappeared. The patient was treated with glycerine and iodine tampons, and hot-water douches, as well as medicinally. She was unable to be out of bed for even a very short time. She menstruated twice while under observation, each time with intense pain--an exaggerated form of the ordinary pain—accompanied with vomiting, excitement, and sleeplessness. After persevering for six weeks with this treatment, I could observe no improvement except that the exudation in the pelvis began to permit more movement of the floor, and a tumor could be less indistinctly made out about the left broad ligament. This I concluded was formed by a Fallopian tube distended with pus. During the six weeks of treatment the patient menstruated twice, each time with the greatest disturbance to the ordinary state of health, including increased pain, vomiting, and headache. Instead of going home to resume an invalid life, the patient resolved, after consultation with her friends, to submit to operation, which was proposed to her. At the operation I found the omentum adherent to the bladder, so that it had to be ligatured and divided before I could reach the tumor. It was then discovered that the uterus was like a small projection in front of and between two bulging tumors which filled up the greater part of the pelvis. With great difficulty the adhesions of the tumor on the left side were partly broken down, and while I was working at the lowest and posterior part of the mass, trying to make a sort of pedicle, the cyst burst and filled the field of operation with a flood of stinking pus and venous blood. The cyst was got away, and after repeated irrigation, sponge-packing, and finally the application of perchloride of iron, the hæmorrhage was arrested, but this was done only after considerable loss of blood, much exposure of the parts,

and the loss of much time. The cyst on the right side was larger and not so tense, and as it was clear that the patient could not stand a repetition of the proceedings, I resolved to stitch it into the abdominal wound. This was partly done, and the tumor was then cut open. Its contents were also offensive pus, which was got away by sponging and irrigation. The pelvic cavity and the whole field of operation were carefully cleaned by irrigation with saline solution. The pelvis was drained with a Kœberlé's tube, and the cyst with a rubber tube, and then the wound was closed. The patient seemed at death's door for about thirty hours. She then began to revive. When she was apparently out of danger, she complained of an intense pain in the pelvis after a violent attack of coughing, and she sank rapidly and died. Examination of the wound showed that the stitched-in Fallopian tube had given way to a slight extent, but sufficiently to permit the entrance of the antiseptic fluid used in dressing into the pelvic cavity. In this case the left cyst was an abscess of the ovary, and the Fallopian tube enlarged, hard, and sinuous, could be felt firmly tied down in front of it, as it were, along the top of the broad ligament. The tube was let alone, as I have repeatedly seen perfect restoration to health in cases requiring abdominal section where such adherent non-distended tubes were not interfered with.[1] The cyst on the right was the Fallopian tube.

As far as the patient was concerned, we had here a frightful state of matters produced by a virulent attack of gonorrhœa, the relation of cause and effect being clear and unequivocal.

Pathologically, was it not a case of suppurative destruction of the appendages? the ovary a mere abscess, the other tube, from one end to the other, an abscess, and the corresponding ovary buried beneath it. The thick-walled cyst is in my possession. Must I look upon it as a sort of sandwich consisting of two layers of specifically inflamed tissue, a mucous and a serous, holding between them a layer of non-specifically inflamed muscular tissue, as Sänger appears to allege? If so, what is the theoretic or practical value of such a view?

Anticipating here a point with regard to treatment, I cannot help thinking, from my experience of many similar cases, that is, of abscess of tube or of ovary *adherent to the pelvic floor,*

[1] See *Medical Chronicle*, November, 1887, and January, 1888.

that the best treatment is by operation from the vagina instead of by abdominal section and the breaking down of the barriers raised by nature against the further invasion of the peritoneum by the process of inflammation. I have had several cases within the last two years which show in a striking manner the efficacy and safety of vaginal incision with drainage.

Effects upon the Ovaries.

Continuing the consideration of the anatomical changes which produce the clinical signs and symptoms of gonorrhœa, we now come to the spread of the infection to the ovaries.

We have seen that great changes may be produced upon the tissues of the Fallopian tubes, resulting in the closure of the ends of their canals and the formation of hydro and pyosalpinx. The fimbriæ not only become matted together by the inflammatory process, but they frequently become attached to the surfaces of the ovaries, and the parts become so modified by the disease in the course of months and years that when they are removed by operation, for the relief of the sufferings caused by the anatomical changes and interference with function, it may be impossible to identify in detail the parts belonging respectively to tube and to ovary. If the disease reaches the fimbriæ, it almost certainly goes on to produce perimetritis and ovaritis. There is no leap from one point to another; the susceptible tissues are continuous. It is the custom to speak of an interval between the ends of the tubes and the ovaries, and to speak of the acute affections of the tissues of the intervening space as peri-oöphoritis. Anatomically there is an interspace; physiologically there is none; and clinically there is no distinct form of disease arising from the inflammation of the particular portion of the peritoneum about the ovary. There is no peri-oöphoritis without inflammation of the ovary itself, and more or less extensive perimetritis; and the symptoms arising from this extension of the inflammation are the symptoms due to inflammation of the pelvic peritoneum and of the ovary. Olshausen, who devotes part of a chapter to peri-oöphoritis, finishes his remarks by admitting that no such disease is distinguishable from pelvic peritonitis. He says: "The symptoms of this peri-oöphoritis differ in no

respect from those of a circumscribed peritonitis. . . . What I have here called peri-oöphoritis may be, in the majority of cases, just as well designated perimetritis."[1] It is, in fact, not in the majority of cases, but in all, just the inflammation of the peritoneum within the area that the ovaries can reach in their physiological movements, and it is, therefore, the inflammation on which the adhesions ultimately formed by the ovaries depend. This fact, perhaps, justifies a distinct name.

Resisting the temptation to paraphrase the usual writing of the text-books on the inflammation of the ovaries, what I have to say on the effects of gonorrhœa upon these organs, whether correct or not, I can concisely state. The inflammation is for the most part superficial. It is a peritonitis rather than a process affecting the substance of the ovary, but that it also not infrequently is a parenchymatous inflammation is proved by the symptoms, by the acute enlargement of the ovary, and by the remote effects. The symptoms of the first acute attack may include the peculiar pain like that with which we are familiar in cases of orchitis, and we may be able, either before the peritoneum has lost its limpness or after the perimetritis, when its subsidence is rapid, to make out the enlarged ovary as an element in the case, but I question very much if this is not the exception. In some severe cases the parenchyma is certainly involved, and the process goes on rapidly to the formation of abscess; but there is then, as has already been remarked, probably some favoring circumstance, such as the pre-existence of some cystic degeneration in a part of the ovary. A case which may be referred to in illustration of this point was mentioned by Dr. Lewers, at a meeting of the Obstetrical Society on January 4th, 1888.[2] It was that of a woman who was admitted into the London Hospital suffering from parametritis a few days after her confinement. She died, and on post-mortem examination it was found that the right ovary was greatly enlarged, "it was adherent to the adjacent surface of the broad ligament by recent lymph, and on section an abscess containing half a drachm of pus was found." The broad ligament itself was as yet only in a state of phlegmon. Thus the ovarian abscess was premature; it

[1] "Die Krankheiten der Ovarien," Stuttgart, 1877.

[2] *British Medical Journal*, January 14th, 1888, p. 78.

was in advance of the general process of inflammatio Probably there was a small pre-existing cyst.

The enlargement which we find later in the history of case is a secondary result, as I shall try to show. By the peritonitis which attacks the covering of the ovary, that covering, the tunica albuginea, is permanently injured. The acute inflammatory state, here or elsewhere, is followed by one of two results: the surface becomes adherent to another surface, or, remaining free, it becomes entirely changed in character—a sort of cicatricial tissue, thickened, toughened, and devoid of its columnar epithelium. There can be little doubt that in every case there is an exudation from the inflamed surfaces, and if this fluid could be imagined, in the severer attacks, as drying up, like the sap of some plants after injury, that is to say, becoming organized into a tough pseudo-membrane, embracing ovaries, tubes, and uterus, we would seem to have an explanation of a not uncommon effect. In some of the cases which we operate upon we may have diagnosed enlargement of the ovaries, dilatation of the tubes, or some other condition accompanied with symptoms which justify surgical interference. On making an abdominal incision we find that by reason of this membranous product of inflammation the examining finger seems to be almost as far from the affected organ as it was in exploring per vaginam. Now the difference between such an extreme case and the mildest, as far as the changes in the tissues are concerned, is one of degree. After an attack of medium severity the ovary becomes in part adherent, and in part the surface is thickened, or covered with a membrane different altogether in character from its original tunica albuginea. The most important consequence of this change appears to be that although the ovarian stroma is little affected, and the Graafian follicles mature as usual, they do not burst externally to the ovary. Where the surface of the ovary is adherent to the bowel, or uterus, or broad ligament, or pelvic peritoneum, the follicles cannot burst; this is obvious enough. But where the surface of the ovary is apparently free they do not burst. A crisis comes in the development of the follicle, and a change takes place in it, but that which would have been a corpus luteum under normal conditions will be found after the same lapse of time a mere apopletic cyst. Such cysts grow. How they

increase I do not know; perhaps by affording a more favorable surface for other follicles to burst through; perhaps by hæmorrhages during subsequent menstrual periods. I have many a time seen such cysts the size of a pigeon's egg, and once or twice even as large as a hen's egg. I do not say that in all these cases the origin of the disease was gonorrhœal, but in all there were signs of old peritonitis, with thickening of the covering of the ovary, and with an entire absence of the signs of recent ovulation near the surface. When this thickening of the covering of the ovary occurs, we can see how certain it is to be accompanied with clinical signs. When the periodic congestion returns, the organ cannot expand as it does under purely physiological conditions. What part of the pain is caused by a sensitive organ pulling upon the adhesions, it is not possible to say, unless the same cause be proved to act, and its amount be ascertained, in the intervals; but that most of the pain is caused by the compression exercised by an unyielding capsule there can be little doubt. The first sufferings caused by the acute onset are those of an attack of peritonitis; they can be relieved, and will subside; but the pain produced at a later period by the efforts of the ovary to expand, or relieve itself from adhesions, is an ever-recurring form of suffering. It may bring on a variety of secondary symptoms, or become so urgent, merely as pain, that operative treatment may be the only mode of giving relief. When the process of disease has reached this stage, the danger to life may be unimportant, but the misery produced by the pain and disturbance of function may be intolerable.

Abscess of the ovary is not one of the common results of gonorrhœa, but that it may occur as the direct consequence of infection there can be no doubt; and it is therefore to be kept in mind as one of the complications by which an attack of gonorrhœa in the woman may lead to a fatal result. Having occurred, it is about as dangerous as pyosalpinx, but it is by no means such an ordinary result of the disease. The great majority of cases of abscess of the ovary are puerperal, or they arise in apparently healthy girls from sudden suppression of the menses, or they occur in scrofulous and tubercular subjects. Gonorrhœa probably results in abscess only in very violent attacks, or when some pre-existing pathological state of the ovary, such as a cyst near the surface, is favorable to

its development. To what extent there is a "mixed infection" when acute ovarian abscess occurs, we have as yet not sufficient clinical material for forming a judgment.

The possibility of an ovary or tube become cystic in the way I have just described, by a unilateral attack of perimetritis, permitting impregnation to occur by means of the healthy side, and ultimately becoming the seat of fatal puerperal septic abscess, has been suggested in a recent paper by Dr. Grigg.[1] The four cases described formed nearly the whole of the mortality of Queen Charlotte's Lying-in Hospital in one year; the patients were all unmarried women of questionable antecedents; and careful post-mortem examination in every case showed that the fatal result was owing to old inflammatory disease of the pelvic viscera, very suggestive of the changes produced by gonorrhœal infection. But for the post-mortem examinations the cases would have been, in all probability, set down as simple puerperal sepsis originating at the time of parturition.

Sterility a Consequence of Gonorrhœal Infection.

We speak of the physical suffering resulting to women from the gonorrhœal infection under various names, implying greater or less structural changes in the internal sexual organs. We say one suffers from leucorrhœa, another from dysmenorrhœa depending upon changes in the position of the uterus, contraction of the canal of the uterus, disease of the tubes and ovaries, or some other cause; another is menorrhagic, and another shows symptoms of one of the more serious affections of the tubes or ovaries, and so on. But, perhaps, the most important consequence may be summed up in the word *sterility.* This is by far the most common result of one or other of the anatomical changes produced by the gonorrhœal infection. *A woman who has suffered from gonorrhœal perimetritis is barren.* Sterility is the result of one or more of the anatomical changes which have been referred to, and co-exists with one or more of the groups of symptoms which we designate by various names, as if they

[1] "On the Dangers arising from Diseases of the Uterine Appendages in Childbed." *British Gynæcological Journal*, Part VII., November, 1886.

were distinct diseases. It is easy to see how the inflammatory process, resulting from the gonorrhœal infection, should have sterility as one of its consequences, whatever other clinical signs or symptoms of disease might be produced. My belief is that perimetritis of both sides most necessarily be followed by sterility. In this condition we see the possibility of fruitful intercourse being attacked and destroyed; the ova are hindered from even leaving the organ which is their source, and, consequently, their impregnation by the male element becomes an impossibility. Even in cases which do not extend to perimetritis, we can conceive that the condition of the Fallopian tubes might be rendered such as to make sterility an infallible result. The tubes become sealed at either end as the direct consequence of the inflammatory process, and even when the canal remains pervious the lining epithelium must be so injured or destroyed that the passage of the ovum is prevented. In such cases of comparatively slight damage, there is danger of the congress of ovum and sperm resulting in tubal pregnancy, or some other form of ectopic gestation.

Should the ova escape from the ovaries, and the tubes remain pervious, there are still the changes in the uterine lining, and in the calibre of the canal, to interfere with the normal course of the impregnation and the development of the ovum in utero. The secretion from the uterine mucous membrane may be so changed as to act as a poison to the ovum, on its arrival in the uterus; but it must be confessed that this condition is more likely to result from a syphilitic taint than from gonorrhœa. The mucous membrane of the uterus may be in such a condition that every attempt at nidification is rendered abortive, or as a somewhat later consequence, stenosis of some part of the canal may occur and interfere mechanically with the passage of the spermatozoa. The usual seat of contraction is the internal os. I am quite well aware that there is a fashion with some therapeutic nihilists among the gynæcologists of sneering at the mention of mechanical obstruction to the passage of spermatozoa as a possible cause of sterility. Being microscopic objects, to them a needle's eye would be a wide thoroughfare. That is clear enough as a question of proportion between the size of the spermatozoon and the diameter of the eye of a needle, but there must be something more in the question than a simple arithmetical

relation. From the nihilists we have not yet obtained a rational explanation of the cases in which dilatation of the uterine canal is directly followed by conception, after years of barren conjugal life. Even if it be alleged that there is a dammed up and changed secretion in the uterine cavity which is the direct cause of separating ovum and living sperm, still the stenosis is the cause of the retention of discharge, and therefore the cause of the sterility.

The stenosis may be secondary, and come on long after sterility has been established by gonorrhœal perimetritis. I have under treatment at the present time a woman of twenty, who has been married two and a half years. There is a clear history of gonorrhœal infection during the first year of married life, and when she came under my care, for the first time, a year ago, there could still be made out some thickening in the perimetrium. There was also a profuse uterine discharge, but the gonococcus could not be detected in it. Since then well-marked stricture of the internal os has come on, with intense dysmenorrhœa of the mimic labor type. A long probe, with a head about half the size of that of the ordinary sound, passes with some hesitation, causing a distinct type of pain, which the patient says is similar to that which precedes and accompanies the menstrual flow. I do not doubt but that this patient will at some future time, in some other hands, be confidently treated for simple stenosis, for the relief of dysmenorrhœa and sterility. The pain may be then relieved, as it will now be by me; the dysmenorrhœa may be temporarily cured, but the sterility never.

The sterility which results from gonorrhœal infection may be produced by the first acts in consummating marriage, as in Case 2, page 271; or pregnancy may occur and end in abortion produced by gonorrhœal disease of the uterine mucous membrane, as in Case 9, page 277; or pregnancy may go on to full term, and be followed by a gonorrhœal puerperal illness with perimetritis. This perimetritis then produces the anatomical changes on which life-long sterility depends. An example of this class, one-child sterility, we have in Case 3, page 272. Whether abortion is ever produced by a gonorrhœal disease of the fœtal or maternal coverings of the ovum cannot be as yet said to be proved, but there is ground for a strong presumption in favor of the existence of a gonorrhœal abortion, and a

sterility post abortum gonorrhoicum. The sterility produced by disease developing in the woman directly after marriage is a familiar and common occurrence. Every practitioner of experience must be able to call to memory cases of the kind, and those who have not been disposed to give to gonorrhœa its just share of the blame, will find, if they carefully investigate the possible causes of sterility in women who were apparently robust at marriage, that the more closely the subject is examined the greater are the dimensions which gonorrhœa assumes as a factor in the production of sterility in the female.

Gonorrhœa is also the cause of sterility in the male in some unknown proportion. Gross inquired into the facts of 192 cases of sterile marriage, and found that the male was the cause in 33 cases, or 17 per cent. of the whole, and that azoospermia existed in 31 of the 33.[1] Kehrer's cases, which are included in these figures, gave 14 in 40 of male sterility, all owing to azoospermia, and in the majority gonorrhœa was shown to be the original cause of the pathological condition. Sänger states that he has found one-third of all sterile marriages to be owing to azoospermia in the male, the result of gonorrhœa. "If we consider, in addition to this, the cases in which sterility is caused by gonorrhœal disease in the woman, the man retaining his potency, we find that gonorrhœal infection takes the first place in the etiology of female barrenness, and is of far more importance than all the other causes put together."[2]

While on the subject of sterility, I have only to add that cases are not unknown in which the gonorrhœal infection sets up perimetritis, and yet the inflammation remains entirely confined to one side. We have an illustration of this in Case 13, p. 303, quoted from Dr. Angus Macdonald's paper. In such cases the women may go on bearing children, just as women do with one ovary removed, as after ovariotomy; but such cases are rare. It is but seldom that the inflammatory process produced by the spread of the micro-organisms remains circumscribed. Either as the result of a continuous process of

[1] J. Matthews Duncan, *Gulstonian Lectures*, 1883.

[2] "Ueber die Beziehungen der gonorrhoischen Erkrankungen zu Puerperalerkrankungen."—*Verhandh. d. deutschen Gesellschaft für Gynäkologie*, Leipzig, 1886.

development the disease reaches the other side of the pelvis, or there is a sudden invasion at some of the early menstrual periods after the infection. A perfect example of this class of case is mentioned in Mr. Lawson Tait's "Diseases of Women" (p. 211).

CASE 22.—"Some years ago a gentleman, who had been a short time married, visited a neighborhood where he unfortunately met a friend of his bachelor days. Within 48 hours he came to me in terrible distress, with the initial symptoms of gonorrhœa, but with the still more terrible dread that he might have conveyed it to his wife, for intercourse had taken place a few hours before his symptoms appeared. Of course, I at once cautioned him to refrain absolutely from intercourse with his wife, advice which I have no reason to believe he disregarded. His gonorrhœa proved very trifling, and passed off entirely in less than a week. Wishing to take his annual holiday he brought his wife to me to make sure that she was free from disease, and I could not find the slightest trace of vaginitis. I therefore sanctioned their travelling to a considerable distance. But within three days I was summoned to her, and found her suffering from a most severe attack of inflammation of the left ovary. After some weeks she got well, though the ovary could be felt, both by rectum and vagina, as large as a small orange, firmly fixed and exquisitely tender. Suddenly the right ovary became similarly affected, and after a most severe illness, during which she seemed frequently at the point of death, she recovered, with the right ovary similarly large and fixed. She never menstruated after this second illness, and she now lives a semi-invalid life, hardly ever free from pain, and unfit for any great exertion, though, as time goes on, her sufferings seem to obtain slight amelioration. She is quite unable to endure marital intercourse."

Whatever exceptional cases we may meet with of women bearing children after an unequivocal attack with pelvic gonorrhœal complications, still we may hold firmly, for all practical purposes, to this conclusion, that *the woman who has suffered from gonorrhœal perimetritis is barren.*

GONORRHŒAL PUERPERAL FEVER.

As to the extent to which gonorrhœal puerperal affections occur there is a great difference of opinion, partly because

there is a want of sufficient observed facts to compel unanimity. Sterility after one pregnancy, whether ending in abortion or full-time parturition, is a fact of frequent occurrence. In all such cases efficient observation would have revealed the presence of puerperal inflammation of the uterus or adnexa, or both. The question is, in what proportion of the cases is the gonorrhœal infection the efficient cause of the barrenness, and in what proportion does the condition depend upon a puerperal septic inflammation? Sänger professes to have already succeeded, by some process of clinical observation, apart from investigation for the presence of micro-organisms, in differentiating the gonorrhœal from the other cases. He has found a type of puerperal fever in which the symptoms come on late in the puerperium. This is his gonorrhœal form; and he maintains that it occurs frequently in women who have been infected with gonorrhœa. "In the gonorrhœal post-partum affection it is the salpingitis which secondarily produces the affection of the pelvic peritoneum, and it does so with a frequency which does not occur in puerperal infection, and also in the pre-antiseptic period could not have occurred. The septic-puerperal salpingitis to which Buhl, E. Martin, Spiegelberg, and others attached so much weight in the causation of puerperal peritonitis, is met with almost exclusively only on the post-mortem table. The gonorrhœal tubal disease of lying-in women leads, perhaps, never to a fatal end, and, after the resorption of the accompanying pelveo-peritonitic exudation, it can usually be readily made out by the bi-manual examination. From this it follows that if there is present a disease of the tube, of the uterine appendages, of the pelvic peritoneum after confinement or abortion, there is reason to suspect a gonorrhœal origin to the ailment. I have been able to find among 230 cases of gonorrhœal infection 35 cases, equal to 15 per cent., in which the gonorrhœal affection of the uterine adnexa and of the pelvic peritoneum could be referred back to the puerperal state, and in the majority of these cases the acute symptoms appeared for the first time six or eight weeks post partum or post abortum."

Sänger may be right in his observations and conclusions. From the present state of our knowledge of the subject, I suppose no one is in a position to assert dogmatically that he is wrong—altogether wrong. I can only say that his state-

ments do not agree with anything I have observed or anywhere read; and there seems to be nothing in the nature of the case to require the symptoms in the puerperal gonorrhœal infection to be always late in showing themselves. We know that in ordinary non-puerperal cases the spread of the infection is often very rapid, and it is hard to believe that a coarse process of washing away by the lochia is one which would greatly retard the development of the mciro-organisms in an otherwise favorable soil. So confident is Sänger in the position he takes up that he goes the length of referring to Angus Macdonald's cases in order to say that the diagnosis was wrong. They were, it seems, not cases of gonorrhœal, but of septic puerperal infection. It is an easy method of supporting a theory; still the conclusion, if true, would have to be accepted. The argument would, however, have been weightier if the author had seen his way to attacking the observations and conclusions of some of his living German contemporaries, who would have been obliged to reply. As the matter stands no reply is possible except from the candid reader; and whoever will take the trouble to refer to the cases (*Edinburgh Medical Journal*, June, 1873) cannot fail to be impressed with the clearness with which the sequence of events is stated, or to be convinced that the diagnosis, made by the experienced and scientific physician who knew all the facts, was in every case correct.

Kroner, of Breslau, has also investigated the relations of gonorrhœal infection to puerperal fever, and reached conclusions widely different from those of Sänger. He made a careful examination of women who had borne children that became affected with blennorrhœa of the conjunctiva, showing the presence of gonorrhœa in the puerperium. He found that 75 out of 82 mothers of children affected with ophthalmia neonatorum passed through a normal puerperal state both early and late. Only one woman, who became affected fourteen days post partum with a severe form of septicæmia, died in childbed, and her illness had no causal relation to the gonorrhœa. Two had a tedious illness beginning with fever soon after the confinement, and only four had any special troubles in the later period of the puerperium. Examination of these revealed the existence of para and perimetric "cica-

trices." Thus Kroner found a comparatively rare concurrence of gonorrhœa and puerperal illness.[1]

In a paper more recently published[2] he maintains that he has obtained still more evidence in support of his original position, and gives details.

I have purposely noticed this subject at a length somewhat out of proportion to the rest of my essay, although it is still almost purely controversial matter, because it seems to me to be a new and important subject for further observation. My own notes bearing on this question must be reserved for the present; they are as yet too few and inconclusive. I am, however, disposed to agree with Noeggerath that most of the cases of salpingitis, which we have been accustomed to look upon of puerperal septic origin, must be classified as gonorrhœal. A peculiarity of this form of so-called puerperal fever is that the patients recover very slowly, and a large percentage of them remain invalided for years, because of pelvic adhesions and deformities, whereas women who have survived an attack of genuine puerperal sepsis or sapræmia pass with comparative rapidity into robust health.[3] There is, unfortunately, too much of a polemical element in the German contributions on this question. It suggests rival schools rather than earnest laborious research with scientific truth only in view. Independent unemotional observation towards the elucidation of the points yet in obscurity is a desideratum.

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[1] "Ueber den Sitz der gonorrhoischen Infection beim Weibe." von Dr. Steinschneider in Franzensbad.—*Berliner klin. Wochenschrift*, April 25, 1887.

[2] "Ueber die Beziehungen der Gonorrhœ zu den Generationsvorgängen."—*Archiv für Gynäkologie*, Bd. XXXI., Hft. 2.

[3] "Ueber latente und chronische Gonorrhœ beim weiblichen Geschlecht."—*Deutsche med. Wochenschrift*, December 8th, 1887.

CHAPTER VI.

TREATMENT, PROPHYLAXIS, AND CONCLUSION.

TREATMENT.

THE treatment of gonorrhœal disease in women may seem, in view of all that has been said, a rather large and complicated subject for exposition. But if we set aside mere theoretical details, and deal with that which concerns only cases met with in practice, the varieties of treatment that can be reasonably employed shrink into comparative insignificance. As a specific disease there is, as a rule, little or no scope for treatment of it in the female; we have only the opportunity of trying to minimise the consequences.

Gonorrhœa as a local disorder requires topical treatment; as a specific disease, depending for its development and symptoms upon the growth of a micro-organism, it requires for its topical treatment the employment of a germicide, one or other of the antiseptic and astringent class of chemicals, which are known to destroy the ordinary micro-organisms.

In women the urethra and bladder become affected as in men, and the disease may spread from thence to the ureters and kidneys. The treatment of this phase of the disease must be practically the same as in men. Now it has been quite recently proved again by an efficient observer that the balsam of copaiva is about the best internal remedy. It was found by Dr. Oscar Oppenheimer, of Heidelberg, that applied directly to the micro-organisms it had little effect, but the urine of a person who had taken the balsam internally acted as a poison to the bacteria.

Oppenheimer[1] experimented with a large number of substances, testing the antimycotic power of the individual

[1] "Untersuchungen über den Gonococcus (Neisser)."—*Archiv für Gynäkologie*, Bd. XXV., Hft. 1.

chemical or drug by cultivation experiments with the coccus-containing matter. Perhaps the most important result of his laborious investigation is that already mentioned with regard to the effect of the decomposition products of copaiva balsam.

Professor Isidor Neumann, of Vienna, has recently published in the *Wiener med. Blätter* some chapters of a forthcoming work on the venereal diseases, and there he calls attention to the danger of kidney disease involved in an attack of gonorrhœa. Now if the danger of kidney disease be as urgent as Neumann asserts, and if Oppenheimer's observations and conclusions are correct, then the rational treatment of urethral and cystic gonorrhœa in the female, as in the male, would be to keep the urine impregnated with the balsam, or that which results from passing it through the body, so that it might act as a germicide, and prevent the invasion of a vital organ by the micro-organisms. It is surprising in the face of these observations to find Dr. Fritz Levy,[1] of Copenhagen, objecting to the use of copaiva in the treatment of women, apparently on the trifling ground that it may cause gastric derangement or a cutaneous eruption.

Along with a suitable internal medicinal treatment there can be little doubt that the best local treatment is antiseptic, and there is a consensus of opinion in favor of the sublimate, although it has disappointed expectation to some extent. Dr. Ulisse Malusardi[2] has lately published a paper, in which he passes in review the history of the therapeutics of gonorrhœa in England, France, and Germany, and gives the results of his own observations and experiments; and he concludes that the sublimate is the most efficient and safest of the antiseptics, while in the later stages of topical treatment the sulphate of zinc may most advantageously be combined with the sublimate.

These facts chiefly bear upon the urethral form, but they may also apply to the treatment of that phase of the disease which we now come to consider, viz., the vulvar and vaginal stages of gonorrhœa in the female. Here I must confess that to me it is a matter of almost purely theoretic speculation. I have seldom seen a case of gonorrhœa in a woman in which

[1] "Om den 'latente' Gonorre hos Kvinden."—*Hospitals-Tidende*, Jan. 19, 1887.

[2] "Studio etiologico e terapeutico sulla blenorragia."—*Gazetta medica di Roma*, May, 1887.

the disease had not already reached the uterus, and I am disposed to think that this has not arisen from want of clinical experience, but from the nature of the facts. We are called upon to treat only the advanced stages. One would expect an opportunity of averting the disease under the circumstances of Case 16 (p. 346), for example; but the opportunity is almost invariably denied. The guilty husband hopes for the best; and believing that if only he can deny and conceal, the wife, even though affected, cannot suffer much harm, and can never be quite sure of the facts, whatever her suspicions may be, he always puts off taking the steps which would procure suitable treatment until some serious symptoms set in. I do not know what may be the experience of those who officially examine professional prostitutes, but I believe decent women in this country who become the subjects of gonorrhœal contagion seldom or never obtain suitable treatment in time. What then are we called upon to treat? Let us take two examples in cases which have come under my notice within the last few months, in order to fix attention upon this point and once more upon the cruelty and ignorance of the gonorrhœic seducer:—

Case 23.—Quite recently I saw, in consultation with a gentleman practising in one of our suburbs, a delicate young lady who was about to be married, but had been compelled, unfortunately, to give up her preparations for marriage, and take to bed on account of some serious illness, supposed by herself and friends to be "inflammation of the bowels." At the time I saw her she had been almost entirely confined to bed for several weeks. Her female relations knew that the illness seemed to begin with profuse and prolonged menstruation, and this surprised them very much, as their young friend had, as a rule, menstruated in a slightly irregular and scanty fashion. The doctor in attendance told me, before visiting the patient, about the state of matters in the pelvis, whose existence we verified together. The patient was feverish, and appeared to be ill. On examination we found that there was an entire absence of the usual signs of virginity; the uterus was large and fixed. There was a great mass of exudation on the right side, some exudation behind filling up Douglas's space, and an indefinite fullness, without so much hardness, on the left side. This state of matters led me to inspect the

vulva, and I found the orifices of the ducts from the glands of Bartholini highly inflamed, and distinctly tender to touch as compared with the surrounding mucous membrane. The discharge from the genital tract was profuse and yellow. My colleague and I were agreed as to the anatomical condition: the only question was as to the origin of the disease, because of its bearing on the prognosis. I expressed to my colleague the belief that the disease was gonorrhœal, and I then learned that such was his suspicion. He had cautiously enquired into the matter, and ascertained that the man whom he most naturally suspected was at the very time confined to bed with some ailment, supposed to be cystitis. Although I could not obtain a satisfactory sample of the discharge for microscopic examination, I do not think there can be any doubt about the diagnosis; the clinical features and collateral facts are quite sufficient. With regard to this case it should be noted that the disease had spread to the perimetrium before medical advice was sought, probably without any consciousness as to cause and effect on the part of the patient, and that among people who would not for a moment hesitate to obtain any medical aid that might seem to them needful. If they do these things in the green tree, what shall be done in the dry?

CASE 24.—A. B., æt. 25, single, dressmaker, a respectable-looking modest young woman, from a small country town, seeks advice on account of intense dysmenorrhœa, which unfits her for work for two or more days every month. It affects chiefly the left side, but for over twelve months it has been gradually growing worse on the right side, which is now nearly as bad as the original seat of pain. From the anamnesis I learned that until two years and a half ago she was a strong healthy girl, who never had a day's illness. Menstruation used to come on from puberty until then with absolute freedom from pain. She then began to menstruate profusely and somewhat irregularly. She had a leucorrhœal discharge, and commenced to suffer at the periods. For a long time the pain was confined almost entirely to the left side of the pelvis, and it came on the day before the flow, and continued to be very severe during the first two days of the menstrual period. Since the first change in menstruation the periods have continued to be profuse, lasting five or six days. The inter-menstrual leucorrhœa has ceased. On making a vaginal examina-

tion of this patient, I found, as I expected, an absence of the hymen and loss of tonicity in the vagina. The uterus was small, with a slight retroversion: the right side of the pelvis was apparently normal to touch, but somewhat tender on deep pressure; the left side was occupied by a hard firm band, evidently an adhesion from old perimetritis; there was pain on pressure upwards in the region of the ovary, but the bowels were so loaded that during this, the only examination, it was not possible to define the position and relations of the parts. The orifices of Bartholini's glands were bright red and sore-looking, while the surrounding mucous membrane was rather anæmic. I told this patient without further parley that two years and a half ago she became affected with the poison of a contagious disease, and was proceeding to give my reasons, when she interrupted me, and asked if she might explain; and then she told her story. About three years ago she was "keeping company" with a young man to whom she expected to be married. His occupation required him to be absent for several months in a large town named, and soon after his return the patient began to have certain symptoms which she described. On telling her young man of her condition he admitted that he had been suffering from a certain disorder, and he appears to have given her the benefit of his experience in treating herself. From her description of her symptoms she went through a mild attack of gonorrhœa, including perimetritis, but she never once consulted a doctor. She solemnly assured me she had lived a perfectly chaste life since her paramour left her, and when she consulted me about the dysmenorrhœa she had no idea that her present symptoms and her former illness were in any way related.

These are by no means extreme cases of gonorrhœal infection in women as it presents itself to the doctor for treatment. But the treatment of such cases is no more treatment of gonorrhœa than the performance of a plastic operation on a contracted cicatrix is the treatment of a burn. We are not treating the venereal disease, and, in private practice, we seldom or never get an opportunity of doing so.

However, if we should meet with cases in the early stage, before the uterus is invaded, we must be prepared to act energetically, and the question arises as to what is best to be done. When men become more impressed with the serious

nature of the disease, it will probably be the rule to see cases in the early stage. Schwarz,[1] of Halle, who speaks so as to give the impression of having had a large experience of purely vulvo-vaginal gonorrhœa, describes his method of treatment in the following terms: "First of all the vulva and vagina are thoroughly cleansed of the adhering secretion by means of a 1:1,000 sublimate solution; then, with the help of a Simon's speculum, the vagina and vulva, including every fold and recess, are energetically swabbed with a dossil of cotton-wool soaked in a one per cent. solution of the sublimate, and rubbed with it for several minutes, so that the superficial sheaths of the epithelium containing the gonococci are removed. The Simon's speculum, or some other with separable blades (such as Bozeman's, &c.), is essential for the purpose in view; by this means it is possible to distend the folds of the vagina to their utmost extent, and to obtain a complete controlling view of the whole process, so as to avoid missing any of the diseased patches. Special care is taken with the introitus, which contains numerous folds.

"The next step is copiously to dust over the vagina and vulva with iodoform, which is still more effectively applied by rubbing it into the mucous membrane with the tip of the finger.

"To complete the process, the vagina is with moderate firmness packed full of iodoform gauze.

"If the treatment is very painful, a thing which depends upon the intensity of the disease process, and the idiosyncrasy of the patient, a narcotic or anæsthetic must be administered.

"The process is of value only when thoroughly carried out, but then it is certain to succeed.

"If, as is usual in rubbing the vagina, there occurs extensive capillary hæmorrhage, it is only a favorable sign, inasmuch as it shows that at the bleeding points the diseased epithelial covering is for the most part removed, and, at the same time, a large number of the superficial, perhaps diseased, capillaries are destroyed.

"The iodoform gauze is permitted to remain for three or four days, and then the whole process is repeated with the same thoroughness, and over the same area.

"After four or five days more the gauze is finally removed,

[1] *Loc. cit.*

and then, for eight to fourteen days, the patient carries out a copious irrigation of the vagina with a sublimate solution of 1 in 2,000."

This process, he says, is hardly ever known to fail. The vagina is red and raw after the second tampon has been removed, and there is usually a copious purulent discharge, "but the gonococci are annihilated, and have for ever vanished."

Schwarz goes on to recommend the use of iodoform to the vulva, in order to eradicate the disease from Bartholini's glands, and he further employs compresses soaked in warm sublimate solution with the same object.

In estimating the value of this treatment, however, it may be as well to recollect that Malusardi's experiments tend to discredit iodoform as a germicide for the gonococcus; and Schwarz is still a believer in a gonorrhœa of the vagina, a phase of the disease whose existence has been rendered still more problematical by some recently published observations[1] made by Dr. Steinschneider, in Neisser's Clinic at Breslau.

The chief objection to Schwarz's method is, however, not so much its inefficiency as its severity. It is clearly unsuitable for even hospital practice in England, and, however efficient it may have proved, it must give way to some milder method.

Fritsch, of Breslau,[2] strongly recommends a solution of chloride of zinc instead of the sublimate. He says he has obtained "quite surprisingly good results." He prescribes a solution of equal parts of water and chloride of zinc. Of this solution about five drams are added to a quart of water. This fluid is warmed and used by means of the syringe twice a day, not leaving off during menstruation. If the simple syringing fails to cure he applies a stronger solution of the zinc chloride to the internal surface of the uterus, and then introduces a pencil of iodoform.

Perhaps a large part of the disappointment with the sublimate, of which so much was expected, arises from an inefficient manner of using it. It partially fails in the gonorrhœa of the male urethra, because it has to be merely injected, and probably forms on the surface of the mucous membrane an albuminate which actually protects the deeper-seated gonococci

[1] *Berliner klinische Wochenschrift*, April 25, 1887.

[2] "Notiz betreffend die Behandlung der gonorrhoischen Vaginitis und Endometritis."—*Centralblatt für Gynäkologie*, No, 30, 1887.

from harm. In the female genital tract, on the other hand, it can be rubbed in, and if the new pathology of gonorrhœa be correct, it ought never to fail.

As to the treatment of the disease after it has reached the uterus, I can speak with confidence. In order to be effective treatment must be energetic and immediate. If we have fair grounds for believing, from the absence of thickening of tissues, or of pain on either side of the uterus, that the disease has not reached the tubes, we may still hope to prevent the worst complication of all. Nothing should be done that could increase the congestion of the uterus and interfere in any way with the uterine canal until the moment the germicide is to be applied. Therefore we cannot stop to dilate with tents; the interference and delay would be fatal to success. Only once, when the hollow sound would not pass, I have rapidly dilated, wounding the mucous membrane, before applying the germicide; and this is an undesirable incident, although it does not necessarily do harm. My routine treatment has been to steady the uterus by holding its anterior lip with a vulsellum, after exposing it well with a duck-bill speculum, and to introduce the full length of the canal a Fritsch-Bozeman sound. The sound is kept in a hot disinfectant, and at the last moment the air in it is expelled and replaced by the ordinary tincture of iodine, which is injected from an ordinary two-ounce glass syringe, connected with the sound by indiarubber tubing. When the sound is in position in the uterus the tincture is slowly injected as long as it continues to flow from the opening in the outer tube of the instrument. It is almost always necessary, however, to withdraw it, in order to clear it of coagulated blood and mucus before we can efficiently complete the application; and this is a serious defect in an otherwise satisfactory instrument. The vagina can be protected from the action of the returning fluid either by rapid and frequent changes of pledgets of cotton-wool, or by constant irrigation by gravitation with warm water.

A strip of lint wrung out of sublimate solution is left in the vagina for two or three hours to give the patient a rest, and then a copious vaginal douche with a weak sublimate solution is used, and this is repeated before the next intra-uterine application. The applications should be repeated three days in succession, if no symptoms arise. The Fritsch-Bozeman

sound is laid aside for several days after the third application, and the vaginal irrigation is continued, careful note being taken of both the axillary and vaginal temperature. When the eschar or iodised uterine lining has had time to break down and get more or less expelled, the process is repeated. So far, I have seen no harm come of the intra-uterine applications, and I am confident that the proceeding has saved several patients from the further spread of the disease. Tincture of iodine is so far the only fluid I have employed. Perhaps a spirituous solution of corrosive sublimate might be used with advantage.

Schwarz employs, at the stage of the disease under consideration, a process of constant irrigation of the whole uterine canal with carbolic acid or corrosive sublimate solution. He mentions some details of three cases in which this method was most successful. The irrigation is carried on for from two to four days through a modified Fritsch's sound, and the return fluid is collected and drained away by means of a special apparatus.

Dr. Fritz Levy, in the paper already referred to, speaks of curetting the uterine canal "without previous dilatation," before applying tincture of iodine or other germicide solution. The treatment seems irrational and barbarous. No good can come of cutting up the endometrium with a curette, which, if so small as to pass the internal os without dilatation, cannot possibly bring away the bacterium-containing débris of mucous membrane which its employment produces. You cannot in this coarse mechanical way get nearer to the gonococci, and you can only succeed in choking up the catheter used to inject the germicide fluid, while laying the tissues open to the further ravages of the disease.

So much, then, must serve here as suggestions for the treatment of the uterine stage. I must confess that the opportunities which have presented themselves to me have been too few for trying variations in treatment. In the vast majority of the cases which I have seen the disease had already spread beyond the uterus by the time the patient applied for advice.

When the disease has advanced beyond the reach of topical treatment, it must be dealt with on a totally different plan. The treatment cannot now be too cautious. Nothing should be done that could possibly increase the blood-supply to the

internal sexual organs, and special care should be taken when the menstrual periods are impending. It should be remembered that some pathological conditions of the ovary may already exist which may greatly exaggerate the effects of the infection. The patient's sexual health is almost certainly about to be ruined, but her life is also in danger. If the ovaries are healthy and the tubes do not become sealed, this danger to life is comparatively slight. If even a small amount of fluid has collected in the tubes it appears to have a tendency to change into pus; and, if the fimbriated end of the tube has not become firmly sealed, undue manipulations setting up strong peristaltic action may produce rupture, followed by a fierce conflagration over the pelvic peritoneum. Such an inflammation, when set up by a fluid resulting from mixed infection, would probably not become rapidly circumscribed, but might be the origin of a general peritonitis.

It is usually necessary to keep down the vaginitis by a *warm* douche, and this is really all the local treatment which appears to be called for beyond poulticing in the acute stage of the perimetritis. The warm water, medicated or plain, may have some soothing effect; the *hot* douche may be injurious. In one of my early cases, which ended fatally (Case 6, page 274), I have always blamed the hot douche for again lighting up the inflammatory process. In order to prevent the uterine discharge from keeping up the disease in the lower portion of the genital tract, I direct the patient, even in the later stages, to use a lotion made by means of tablets of corrosive sublimate and ammonium chloride.[1] Each tablet contains two grains of the sublimate, so that when one of them is broken and thrown into a pint of warm water, the resulting solution is practically the strength of 1 in 5,000. A solution prepared in the same way should also be employed in the earlier stages of the disease, concomitantly with the intra-uterine applications. This treatment is continued until the dangers of the acute and subacute stages are over, and it is time to endeavor by suitable measures to remove the resulting exudation, to break down adhesions, and to prevent deformities.

The remoter stages of the disease, those of complications and consequences, must be looked upon as a separate chapter

[1] These tablets are prepared by Messrs. Wright and Barnaby, chemists, Oxford Road, Manchester.

in gynæcology, apart, to a large extent, from their primary cause. Gonorrhœa in a woman, when it has once passed beyond the uterus, is an incurable disease. Such an outrage as the invasion of the Fallopian tubes by a pathogenic organism Nature never forgives. Just as a burn inflicted on a child that falls in the fire is followed by a deforming cicatrix, the effects of which may be ultimately more or less relieved by surgical measures; so the consequences of gonorrhœal invasion of the tubes and peritoneum are adhesions and deformities which cannot be cured, but whose miserable consequences may be sometimes palliated by operative gynæcology.

Prophylaxis.

When we come to consider the prevention of the disease under discussion, we are met by the question: What is the legitimate function of medicine in relation to the venereal disorders? It cannot be to make vice and excess safe and easy. Medicine is not called upon to become "Procuress to the Lords of Hell." How far it may be legitimately requested to give instruction in prophylaxis, with a view to stamping out the maladies, is a question in ethics for which, fortunately, I am not called upon to offer a solution.

One relevant question, a portion of the subject, presents itself and cannot be thrust aside; and regarding it the reader, who has not given it special attention, may justly claim from the essayist, who has had the temerity to take up the subject, some suggestion, the fruit of his presumed special thought and experience. The question is: How are women to be protected from the consequences of their husband's pre-nuptial or post-nuptial vice and folly? I fear no concise and adequate answer is forthcoming. One of our greatest living philosophers has well said that the abrogation of the sexual relation would eliminate most of the poetry from human life. The destruction of that relation in the individual case, and the substitution of loathing disgust, and physical suffering, at the moment when aspirations, unconsciously the outcome of sexual vigor, which lend poetry and dignity to existence, and make men and women something more than mere mammals, are in process of reaching fruition, is a result peculiarly repulsive,

and productive of resentment against its causes. And we naturally ask: How can the sum of human misery, resulting from this special cause, be diminished? Can the women protect themselves or through their guardians? The less that women know of these matters, and are capable of suspecting, the better for society; and we can hardly conceive of a state of society in which this evil received so much attention that the guardians of young and innocent women usually inquired as jealously into certain details in the antecedents and present physical condition of suitors as they are wont to do now into their worldly circumstances.

Can we expect anything from legislation? Hardly. We have probably gone as far in that direction already as a healthy public sentiment could permit. Some of our legislation has been useless or worse, and has had to be repealed; and while recent legislation for the protection of young women may do good, if the laws be calmly and justly administered, it does not to any considerable extent reach the causes of the suffering with which we are at present concerned. If legislation could do something for the protection of young and thoughtless lads, thrown upon their own resources in our great cities, against the harridans who lie in wait for them, and in modern times perform the functions attributed to the fabled harpies, there might be some diminution in the amount of contagion afloat; but we need not look for such legislation. Men must protect themselves: they must avoid the evil or take the consequences. If the consequences were confined to themselves, nothing more need be said. But the innocent suffer most. Gonorrhœa in the woman is as ruinous as syphilis; there is little to choose betwixt the diseases. The men must protect the women. The great majority of them are amenable to reason, and would listen to the dictates of humanity and of honor if their consciences were appealed to. With regard to their wives, there is also the sentiment of self-interest to influence their actions. It only needs definite knowledge to be diffused among them as to the risk of inflicting untold suffering upon those whom they wish to protect, to greatly influence their conduct; and the only guardians of the necessary knowledge are the medical profession. While the doctors look upon gonorrhœa in women as a mere bagatelle, what can be expected of their self-indulgent male clients? As long as the medical

practitioner believes that he can produce a "precious result" at a critical time with a little nitrate of silver solution, the education of the layman will not begin; he will not be adequately impressed with the physical grounds for exercising self-control in order to escape infection, or for the need of persevering industry and self-denial in order to completely and rapidly eradicate the disorder once contracted.

The attentive reader of the notes of cases given in the foregoing pages will have observed that the worst results followed to the women whose husbands had been the victims of gleet or neglected gonorrhœa even long before marriage. The practical lesson from this observation is obvious enough: (1) Measures should be taken to have the disease cured as quickly as possible on symptoms beginning to appear; and (2) when a chronic disorder has become established it should be a positive bar to marriage until there is sufficient proof that it has been cured for many months.

And what is the criterion of cure? The question is perhaps as difficult to answer as the analogous question with regard to syphilis. We have seen that in some cases, where serious consequences to the woman followed soon after marriage, a slight urethral discharge or moisture reappearing in the male owing to post-nuptial sexual indulgence. Taking a hint from this fact, we may find a criterion of cure in some cases. The local effects of sexual excess may be sufficiently reproduced, in a given case of suspicion, by the injection into the urethra of a nitrate of silver or corrosive sublimate solution, so as to irritate and produce a slight urethral discharge, in the same way as a conjunctival discharge is produced by Credé's method of preventing ophthalmia neonatorum. The urethral discharge resulting should be carefully and repeatedly examined for the gonococcus, and a negative result would afford a strong presumption of cure. The slight sacrifice of comfort thus made by the patient would be too trifling to be considered when the importance of the result was kept in mind. This is far from a perfect method of gaining definite information, but as a criterion of cure it is better than nothing at all.

Sound judgment is hard; and it may be that in writing this essay I am joining the band of those who exaggerate the im-

portance of the form of venereal disorder under discussion, or at least see too much in recent observations and experiments. That such observations and experiments must be continued and greatly extended, sifted, discussed, and subjected to the most rigorous logical processes before we can fill up *lacunæ* in our knowledge, reconcile differences of opinion, and "in its season bring the law," I willingly admit; meanwhile we have learned enough for that practical application of our knowledge which in its turn becomes a method of research and an agent of progress. In putting this subject before the readers of my essay, I do not think I in any way exaggerate; I believe rather that in attempting to depict the fruits of our newer knowledge I use but feeble colors. I believe that the gynæcologist who fails to closely examine into this subject, repulsive though it may be apart from its scientific interest, must constantly fail in his duty to his patients; and I am convinced that when the general practitioner, to whom the domestic *arcana* are an open book, comes sufficiently to observe the phases, and duly to appreciate the import, of a large class of ailments with which he is constantly called upon to deal, the view which I expressed in the commencement of this essay will become the generally accepted opinion of the medical profession, viz., that the gonorrhœal infection in women gives rise to a group of diseases which, by reason of their clinical interest and their social and moral consequences, surpass in importance every other class of affections which claim the attention of the gynæcologist.

ON GIDDINESS.

FROM A SERIES OF

Clinical Lectures on Important Symptoms.

BY

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ON GIDDINESS.

LECTURE I.

GENTLEMEN: We have frequently met with giddiness as a prominent symptom in cases which we have studied in the wards, and I promised that I would take an opportunity of discussing the subject in the lecture-room, illustrating my observations by cases which I might be able to show you or describe. I find it useful, sometimes, to take up a symptom in this way rather than to discuss individual cases or groups of cases, as we usually do in this lecture-room.

Giddiness is a sensation with which we are all familiar, but which it is difficult to describe. Its essential feature is the feeling of uncertainty of our position in space relatively to surrounding objects. Sometimes one feels as if objects were whirling round more or less rapidly; sometimes one seems to be moving in the midst of them; and that apparent movement may be round a vertical or a horizontal axis, to one side or other, forwards or backwards; or there may be a sensation of sinking downwards; or the movement may appear confused or irregular. It may also be slow or rapid.

Giddiness is often associated with other disorders of sensation, such as nausea, deafness, tinnitus aurium, or other subjective sounds, dimness or other changes of vision, faintness, palpitation, hallucinations, and is sometimes followed by loss of consciousness. But it is constantly associated with impairment of equilibriation. This is readily explained when we consider that it is the result of derangement of a function essentially related to equilibriation,—disturbance of equilibriation being, so to speak, the motor aspect of the sensation of giddiness. For equilibriation we require the co-ordinated

action of many groups of muscles. This co-ordination is, like others, guided by sensory impressions, and these impressions are derived from different sources.

Neither the power of equilibriation, nor the power of appreciating the impressions by which it is guided, is inborn in man. Each is gradually developed by processes of education. The senses which are trained to subserve it are touch, sight, the muscular sense, probably an articular sense, and a visceral sense, along with the great organ of special sense for equilibriation—the semi-circular canals. Of some of the impressions we are conscious, of others not. Of sight and touch impressions we habitually take cognizance, or at least we may do so; of the other kinds of impressions we make use without any act of intellectual perception. It is at once apparent how sight subserves the purposes of equilibriation, and it becomes especially noticeable when the other sources of sensory impressions are impaired. You have often seen this in cases of locomotor ataxia. You have noticed how helpless the patient becomes when his eyes are closed. The importance of touch, and particularly plantar touch, is equally familiar to you, being often illustrated in the same class of cases. The impairment of the muscular sense, I have sometimes shown you, is at least as important as those already mentioned. The articular and the visceral are less obvious, but I think are real. I shall not dwell upon them, but the special sense for equilibriation, which has its seat in the semi-circular canals, requires fuller notice.

Let me remind you of some anatomical facts and physiological explanations. The canals are situated in the petrous portion of the temporal bone. They are three in number, and communicate by means of five openings with the vestibule. Each canal has a dilated or ampullary end, and each by its ampullary end opens into the vestibule, while two of them have their non-ampullary ends united, and communicate with the vestibule by a common opening. These canals occupy different planes, and are known as superior, posterior, and horizontal, according to their position. Within the bony labyrinth there is a membranous labyrinth, the membranes being much smaller than the canal, except at the ampullary ends. Between the bony canal and the membranes is the perilymph, with connective tissue, blood-vessels, and nerves. Within the membranous canal is the endolymph. A branch

of the portio mollis of the seventh nerve is distributed to a crescent-shaped ridge near the middle of each ampulla. The terminal nerve-structures are hair-like cells, which project into the endolymph, and which must obviously be affected by changes in it. Through the chain of bones the labyrinth is connected with the tympanum. The handle of the malleus is fixed into the tympanum, while its head articulates with the incus. The latter, again, is connected with the stapes as well as with the posterior wall of the tympanum. The stapes is fixed into the fenestra ovalis, and by its movements produces alterations in the state of the fluids in the whole labyrinth.

In 1828, Flourens discovered that the semi-circular canals have something to do with the maintenance of the equilibrium of the body. He found by experiment that section of a membranous canal was always followed by movements of the head, or even of the body, rotating about an axis at right angles to the plane of the divided canal. On section of the horizontal canals, the animal moved its head rapidly from side to side and tended to spin round on a vertical axis. When the posterior canals were cut, rapid movements backwards and forwards took place, and the animal showed a proclivity to turn somersaults, from before backwards. When the superior vertical canals were cut, similar movements occurred; but the tendency now was to turn somersaults from behind forwards. Section of all the canals induced peculiar irregular movements. The movements always became worse if the animal was excited or disturbed. If one side only was cut, the abnormalities gradually subsided; if the canals of both sides were injured, the derangement of motion was permanent. These were clearly not the mere results of irritation, seeing that the symptoms continued long after the external wounds had been healed.

A great advance was made by Goltz when he suggested, in 1870, that pressure of the endolymph gives rise to sensory impressions, and that, according to the position of the body, the pressure varies by gravitation in different portions of the series of canals. He showed that the sensory impressions bear an important relation to equilibriation, and that by altering these impressions he could produce results similar to those which follow section of the canals. He fixed the heads of pigeons in peculiar positions, tying, for example, the head

down upon the breast, and found that equilibriation was thereby impaired.

The next advance was made by Cyon in 1872, who repeated and confirmed the former observations, and formulated the theory that from the semi-circular canals we derive impressions as to the position of our heads in space, each canal being definitely related to one of the dimensions of space, that these impressions guide us in equilibriation, and that failure of equilibriation may be due to disorder of their functions.

In 1875, Professor Crum-Brown published an interesting account of some experiments which he had made upon the subject. He pointed out that if the body be passively rotated, one can, with the eyes bandaged, determine pretty accurately the amount of movement made; that if such passive movements be continued beyond a certain time, the feeling is lost, but that if the rotation be suddenly arrested, the sensation of rotation continues. He suggested the theory that the sensations are due to alterations of pressure in the endolymph, not from mere gravitation, as had been supposed, but from the formation of currents in the endolymph, that fluid being conceived to lag behind in the rotational movement of the head. He pointed out how well such a theory suffices to explain the facts discovered by himself, as well as those previously known. Moreover, he remarked, that as each canal has but one ampulla, and as the same organ of sense can produce sensations differing in quantity only, and not in quality, it is probable that one canal will respond only to the rotation round one axis, and in one direction, because the sensation of rotation in the opposite direction is qualitatively different. Hence he concluded that the semi-circular canals are paired organs, and that each canal corresponds only to rotation in a single direction. The same theory was promulgated independently, and at about the same time, by Professors Mach of Prague, and Breuer of Vienna.

Some later experiments of Cyon throw doubt upon this kinetic theory, but cannot be held, in my opinion, to have disproved it.

You perceive, then, that we have abundant proof that the semi-circular canals are sensory organs subserving equilibriation, and that we are gradually acquiring definite ideas as to how their functions are performed. Besides these peripheral

structures which guide us in our equilibriation, there are, of course, conducting fibres and nerve centres which subserve the functions. Changes in any part of these structures may suffice to produce the sensory and the motor abnormality. We shall first consider the peripheral causes of giddiness, and then those due to changes in the conducting fibres and nerve centres.

In normal equilibriation we guide ourselves by information derived from all the peripheral sources to which I have referred—and in normal conditions they all harmonise—the information derived from sight, from touch, from the muscular sense, from the labyrinth, all correspond. When they do not, giddiness results. Observe, then, that peripheral giddiness is usually due to *contradictoriness of sensory impressions.*

Sometimes the contradictoriness is due to external conditions. This is especially true regarding sight. If you visit the famous Leaning Tower of Pisa, you will most likely experience, as you ascend and descend its stair, a peculiar, uneasy feeling of slight giddiness. On experiencing this sensation, it occurred to me that it was probably due to contradictoriness of impressions derived from sight and from the other sources of impression guiding equilibriation; and I found that it was so, for, by slightly altering my attitude, the feeling at once disappeared. In the tower there were three others along with me, all of whom felt the same uneasiness, and all were freed from it by altering their posture very slightly. I am satisfied from what I saw when there, and from what I have heard since, that that peculiar giddiness is felt by very many people. Some of you may have visited, and many of you will hereafter visit, the famous tower. When you do so, you will be able to verify what I have told you.

But you have all had opportunity of observing another variety of the feeling—what we may term cliff or tower giddiness. A large proportion of people suffer more or less from an uneasy feeling of giddiness whenever they stand upon a tower or a cliff, or even a steep stair or a hillside. It is due, I take it, to contradictoriness of impressions. They have been accustomed, under ordinary conditions, to receive simultaneously impressions from the plantar surfaces, from the muscular, articular, visceral, and labyrinthine structures, with visual impressions of solid ground before them; but when standing

on a tower or cliff they find the visual impressions contradicting the others, and so the peculiar feeling arises. The liability to this kind of giddiness varies in different individuals, and is determined by the sensitiveness of the nervous system, on the one hand, and by the degree in which the individual has been accustomed to trust to visual impressions for equilibriation, on the other. You will find the feeling aggravated if you look downwards to where, according to experience, the solid ground should be, and absent or diminished if you look in other directions. Some people are so sensitive in this respect that they cannot with comfort sit in a gallery of a church or theatre; some of you may feel that you cannot with comfort occupy the front gallery seat in the new Practice of Physic lecture-room in the University.

A very curious condition, manifestly allied to this, was described a few years ago by two eminent authorities, and is coming to be known to the profession as "Agoraphobia." Benedikt described it in 1870; Westphal in 1872. Its essential feature is, that while the subjects of it can walk quite well in a narrow lane, or even in an ordinary street, they become uneasy, and experience either giddiness or a peculiar feeling allied to it, when they attempt to traverse a wide market-place or square, such as narrow lanes often open into in continental towns.

Let me describe by way of illustration one of Westphal's cases. The patient was a commercial traveller, aged thirty-two, of middle height, slender, healthy-looking and mobile, and complained that he suffered greatly when he tried to walk across open squares; when he attempted to do so, a peculiar uneasiness, situated mostly in the head, but attended by violent action of the heart, was induced. On attempting to cross a square he felt as if it were miles wide, and often he trembled all over; the more he approached to the houses at the side the less was his uneasiness; a stick was no help to him, but walking arm in arm in lively conversation helped him considerably. If absorbed in thought, he could sometimes accomplish the feat; if he kept close to a carriage crossing the square he could manage pretty well; he had to deny himself the pleasures of the Thiergarten, and could not by any effort make his way to Charlottenburg (a public garden and favorite promenade in the neighborhood of Berlin). The same uneasiness

was felt in some measure if he had to walk beside a blank wall or when the shops were shut; he usually supped in an eating-house, and had to help himself home by ingenious devices; he waited till he saw people going in his direction, and followed them up, or he followed closely behind a conveyance. When asked what he would do if left without any of these external guides, he said that the very thought appalled him, but that he would probably throw himself on the ground and hold on by the grass. In his frequent journeys he had constantly to avail himself of stratagems of war, so as to be prepared under any circumstances to get assistance. On entering theatres and churches a similar uneasiness occurred at one time; but that difficulty disappeared. He could sail without much discomfort in a large vessel; not in a small boat; and could only walk at the sea-side in places where the houses come close to the shore. He lived in constant dread of these uneasy feelings. He described them as differing from giddiness and from the feeling experienced when looking from a tower. He benefited by moderate use of alcoholic stimulants. The disease began in his twenty-seventh year. The nervous system was not otherwise abnormal; but there was a slight want of symmetry of the two halves of the body. There was no marked history of nervous disease in his family.

In this patient it will be observed that there was abundant evidence of the peculiarity of the nervous organization, but the point to which I wish to draw your attention is the inability to walk across an open square, and the peculiar sensation experienced by the patient. Benedikt speaks of the sensation as giddiness, and although Westphal has drawn a distinction, as this patient did, between ordinary giddiness and this special uneasiness, I think we are warranted in believing that the feelings are closely allied.

Various explanations of the condition have been attempted, but none of them seems to be quite satisfactory. A reasonable explanation of one element of the trouble lies, I think, in the supposition that these patients, with the nervous system morbidly sensitive, have from some peculiarity acquired the habit of guiding themselves in their equilibriations by reference to near vertical lines; and that, when these vertical lines are wanting, they feel as others do when standing on the edge of a cliff. Notice the fact about the methods they devise for

getting across squares. If they can get behind some vehicle, they can walk well enough; the vertical line of the vehicle suffices to guide them.

I have met with one case of another condition allied to these. I know a lady who experiences great discomfort if she sits in a building with a lofty ceiling. She always requires to sit under a gallery in church; and the explanation is, that to her it is essential that she should have the impression of a plane not far off above her head. In these forms of giddiness you observe that, on the one hand, we have certain nervous susceptibilities more or less pronounced, combining with certain external conditions to induce this sensation. I shall now refer to some causes of giddiness with which you are probably all more or less familiar, as they occur in a large proportion of people when placed under certain conditions.

There is, first, the giddiness resulting from rapid rotation, —the waltz giddiness. This appears to be due, not so much to the contradictoriness of impressions, as to powerful impressions made upon the special centre for equilibriation, the rotation producing changes in the semi-circular canals, well explained, as we have seen, by the kinetic theory of their function. Every one naturally becomes giddy with rapid rotation, and with this, under ordinary conditions, equilibriation fails. But one may be trained to equilibriate, notwithstanding the labyrinthine impression. All physical education consists in learning to co-ordinate muscular movements. In learning to waltz, the child has not only to learn to co-ordinate certain groups of muscles, but has to learn to do so while he is receiving misleading impressions from sensory organs on which he is accustomed to rely. He has to guide his movements by the plantar, muscular, articular, and other impressions, and to time them by the music, disregarding his labyrinthine impressions altogether.

Swing-giddiness is not unfrequently experienced by children when they are enjoying that exhilarating amusement. The giddiness you will find, if you analyse it, is due to a variety of causes. There is obviously the constant changing of the visual impressions. But this is not all. The feeling is most marked when the swinger is descending. Now, during the descent, a very peculiar visceral sensation is experienced. It appears as if the body, as a whole, descended at a greater rate than the

abdominal viscera, and as if they followed; and as they followed, a certain friction took place between the peritoneal surfaces—the visceral sensation being attended by a quite peculiar uneasiness. It is probable that, in the cranium and in the semi-circular canals a similar action may take place; but these we cannot so readily determine as the visual and the visceral impressions.

In sea-sickness, with its attendant giddiness, we find many elements contributing to the result. There are visual impressions—some very susceptible persons becoming affected even when they look at the sea; but every one who suffers from it feels his discomfort increased if he watches the varying elevations of the bulwarks of the vessel and the surrounding waves, and feels the better of looking another way, or of closing his eyes. But in this condition also the visceral impressions are most important. As the vessel sinks into the trough of the wave the body seems to descend as in the swing, the viscera appearing to sink downwards at a slower rate than the body generally. And here, as on the swing, this is attended by a terrible uneasiness. I suppose that one of the great advantages of the sea-sick invalid laying himself down at full length is that he thereby diminishes the visceral movement. With the labyrinthine impressions also the nerve centres must be sorely puzzled, for from moment to moment the impression varies as the vessel rises or sinks, or rolls from side to side. The muscular sense, also, is brought into most unusual conditions, as is also the articular sense. But one other source of sensory impressions is also at fault, for the plantar pressure varies from moment to moment. At one moment, as the vessel sinks into the wave hollow, it seems as if the vessel were falling from beneath the feet, and there is almost no plantar impression; and then, again, as the vessel rises, the pressure becomes greater. In such conditions it is clear that the sensorium receives a great variety of ever-varying and contradictory impressions.

But giddiness may be due to abnormalities of the sensory structures themselves. Notice, first, some ocular causes. In illustration of this I shall recount to you a typical case seen by some of your predecessors. When I first took charge of the clinical wards as professor, I found a patient who was unable to walk or even to stand on account of fits of giddiness,

with which he was frequently seized. On examining him I found no evidence of central nervous disease, but observed that when he took his giddy turns there was always a rapid oscillating movement of his eyes. It occurred to me that this oscillation or nystagmus might cause the giddiness, and I held his eyes fixed by slight pressure. This cured the giddiness in a moment, and while it was kept up the giddiness never recurred. On further inquiry I found that the patient was a miner, and that his case was a typical one of what has been described as miner's nystagmus. The patient continued under observation for some time, but no good was done except by the mechanical treatment.

The giddiness in that case was manifestly not due to any central disease, but was simply a result of the contradictory impressions resulting from the movement of the eyes. The disease is now pretty well known. It is due to spasm from long-continued muscular strain—is indeed a form of trade spasm. The miner while at work lies on his side, and opens up the coal-seam above and beyond his head. He works also in a dim light. He has, therefore, to strain his eyes greatly, and to keep them in an unnatural position, and this nystagmus, with its attendant giddiness, occasionally follows.

You may ask me whether nystagmus always induces giddiness. Certainly it does not. It sometimes is merely associated with giddiness, the two depending upon a common cause, as in cerebellar disease. Sometimes, again, it results from cerebral disease, sometimes from disease of the retina, sometimes from disease of the cornea, occurring in childhood. In such cases, giddiness does not result, because the sight is so much impaired that the patient learns not to be dependent on the visual impressions for guidance. This is especially the case when the cause comes into operation in early life.

Paralysis of the external rectus, when of recent origin, frequently leads to giddiness from double vision; and even if the one eye be closed, a sensation of vertigo is experienced, because the muscular sense contradicts other impressions. You will often see this fact illustrated in the eye wards, but I may mention an example of it which occurred in the case of a patient whose illness I shall describe to you later on in connection with intra-cranial lesions. The patient, a young man of nineteen, naturally of robust constitution and a rather

eminent university athlete, had sustained an injury to the head, and soon afterwards observed that he saw double, and had a constant feeling of giddiness. He soon discovered that if he closed one eye, his giddiness disappeared. It was manifest, then, that the giddiness resulted from the contradictory ocular impression due to squint, which again in its turn was due to paralysis of one of the ocular nerves. The test which I should advise you to apply in such a case is the simple one of getting the patient to close one eye; if this cures the giddiness you may be quite sure that it is of the nature we are now considering. Paralysis of other muscles of the eye may, of course, lead to the same results.

I must defer till next lecture an account of the most important of all the peripheral forms of giddiness, that due to alterations in the semi-circular canals—Menière's Disease.

LECTURE II.

GENTLEMEN: Our last clinical lecture was devoted to the subject of giddiness, and after describing to you the nature of the sensation, I discussed certain of its causes, viz., those due to altered conditions outside the body, and those which result from abnormal peripheral impressions. At the end of the hour I was about to explain the relationship of giddiness to diseases of the semi-circular canals.

Of all the forms of giddiness from peripheral causes, the most interesting and important is that group due to causes situated in the semi-circular canals, which are commonly known as Menière's Disease, or Labyrinthine Vertigo. Dr. Menière is superintendent of the Institution for Deaf and Dumb in Paris, and by the light of the discoveries of Flourens in regard to the semi-circular canals, he concluded that many attacks which had been considered cerebral, were in reality due to disease of these organs. In 1861, he brought his observations before the Academy of Medicine in Paris. As a type of this condition, I shall cite to you one of his most striking cases. A young woman travelled on a winter night on the outside of a stage-coach, and was exposed to great cold. She was at the time menstruating. She suddenly became completely deaf. When received into the hospital wards, the principal symptoms were constant vertigo and vomiting induced by the slightest effort to move. She died on the fifth day. At the autopsy it was found that the brain, cerebellum, and cord were free from morbid change. As the patient had all at once become deaf, although she had her hearing perfect up to the time of the attack, Menière removed the temporal bones, so that he might be able to ascertain the cause of the deafness. The semi-circular canals were the only part of the labyrinth that presented any change; they contained a reddish-colored plastic lymph instead of the fluid of Cotunnius. From such observations Dr. Menière drew his first description of the disease.

That it is not always fatal the first case which came under my care may serve to show. An apprentice boy was brought into my wards suffering from most of the symptoms of Menière's disease, such as vertigo, faintness, vomiting, and reeling gait, and roaring or humming noises in the ears, with deafness of one side. On inquiry, I found that the illness had come on suddenly and been caused by a violent blow on the side of the head which the boy had received when at work. In a few days improvement set in, and in a short time the patient was well. I supposed that, in consequence of the blow, some slight hæmorrhage had taken place into the semi-circular canals, and that the blood so effused was speedily absorbed. Whether this view of the case be correct or not, you will remember that a very speedy subsidence of the symptoms may occur. In some cases, while life is not endangered, the patient continues to suffer for long periods—even for many years—and life is made almost intolerable by the discomfort it occasions.

Some recent writers on the subject, such as Dr. M'Bride, recognize several varieties of Menière's disease. And among these I mention, first, the forms due to *changes in the external ear*. Some people experience all the symptoms of Menière's disease if they have their ears syringed, and often accumulations of wax, or of eczematous secretions, produce the same result, if they be so situated as to press with force on the drumhead.

I show you to-day a patient who has several times consulted me of late. She is fifty-six years of age, and has come complaining of giddiness, pain in the head, with dullness of hearing, a feeling of oppression within the cranium, and a singing or buzzing sound in the ears. Her hereditary and social history are unimportant; but she has lately passed through very severe trials, to which she ascribes her illness. She has two sets of abnormal sensations, one constant, the other occasional or paroxysmal. The constant feeling consists in noise in the ears, uneasiness in the head, deafness, and general discomfort. The paroxysmal consists in severe fits of giddiness, with palpitation, faintness, and coldness. The giddiness is peculiar, in respect that she feels an inclination to fall backwards; and a friend who accompanied her to the infirmary says that this is not a mere sensation, but that she actually tends to fall back. If she is able to sit down and press the back of her head

against anything, the giddiness is at once relieved. Her ears were carefully examined by Dr. Graham Brown, who found that the Eustachian tubes were not readily permeable, while there was a large mass of wax in the right meatus. On this being removed, the buzzing at once ceased; and during the week that has elapsed now since this treatment was adopted, she has felt almost well, and has certainly had none of her paroxysmal attacks. Here, then, is an example of a group of formidable symptoms depending mainly upon a cause situated in the external meatus.

The second group is that *due to diseases of the middle ear*, which is generally considered to be the commonest cause of the symptoms. As an illustration, I shall cite the case of a patient who was under my care in the Infirmary some years ago, and in which I had the benefit of Dr. M'Bride's advice and assistance. The following account is in the words of that gentleman:—

"W. H., æt. fifty-eight, complained of giddiness and staggering. He is a blacksmith by trade, and exposed to sudden changes of temperature; and to this he attributes, in a great measure, his illness. For five years his hearing has been getting gradually worse. Before the patient applied for advice, he had two very severe paroxysms of vertigo, with an interval of two days between them. What occurred on these two occasions seems to have been that the patient felt intensely giddy, heard noises in his head, and lost consciousness. This happened for the first time on a public thoroughfare, and when he recovered his senses he found himself seated and being attended to by bystanders. He then felt sick, and everything round him seemed to turn (direction not observed). He staggered home, and when he got there vomited and fell asleep. On awaking he felt better, and next day went to work, feeling well. On the day following, however, he had a repetition of the attack. Since then the patient has been troubled with constant giddiness and staggering towards the left. A mixture of iodide and bromide of potassium seemed to have little or no effect on the symptoms. Except for the giddiness and staggering, the patient seemed in good health.

"The examination of the ears gave the following results:—Watch (normally heard at thirty inches) not heard in contact with either auricle. Conversation is, however, fairly well

heard, and the tuning-fork is well heard in both ears by bone conduction. Tympanic Membranes.—Right membrane opaque, with vascular injection along the handle of the malleus. Anteriorly and superiorly there is a dark atrophic patch. Left membrane indrawn, with well-marked folds. Politzer's method only succeeded in forcing air into the left tympanum. After this, inspection showed the corresponding membrane flattened out, the folds having disappeared. Air could only be driven into the right middle ear through the Eustachian catheter, and auscultation during the process revealed a distant gurgle, indicating probably an accumulation of mucus in the pharyngeal orifice of the right Eustachian tube.

"On the first attempt to inflate the tympana by Politzer's method, a very extraordinary effect was produced. As stated, air only entered the left middle ear; but whereas, before the operation, the patient had great difficulty in standing and walking, even with assistance, after the inflation he declared himself greatly relieved, and was able to walk about alone and unsupported. The subsequent treatment consisted in astringent applications to the throat and nasopharynx, which were in a catarrhal state, inflation of iodised steam into the tympana, and occasional use of the air-bag and Eustachian catheter. The result of this treatment was that in a week or two the only symptom of vertigo left was that looking down suddenly produced slight giddiness. The hearing also improved considerably; that is, he could hear a watch, which was previously altogether inaudible, on the right side in contact with the auricle, and on the left just off the ear. The tinnitus also, from which he suffered, was much relieved."

Commenting upon these facts, Dr. M'Bride says: "Now, this case is perfectly typical of what is often known as Menière's disease in an aggravated form. The vertigo, tinnitus, vomiting, and loss of consciousness were all well marked The deafness, however, was most distinctly traceable to chronic middle-ear catarrh, pure and simple. There was no organic mischief in the cochlea, as shown by the tuning-fork test, nor in the semi-circular canals, as proved by the success of local treatment directed to the middle ear. Moreover, we could trace the nervous phenomena to deficient ventilation of the left tympanum; for whenever air entered it there was an immediate and marked improvement. It was on that side, too,

that there was most evidence of altered intra-tympanal tension in the appearance of the drumhead; and we have before seen that changes of tension in the tympanum produce a more or less corresponding alteration in the endolymph. The case is important as one showing the most marked and aggravated characteristics of Menière's disease, depending upon a curable condition of the middle ear—curable, be it observed, not by empirical inunction of iodine and application of blisters, but by rational treatment directed to recognized pathological condition. I have seen a considerable number of cases where staggering and giddiness depend upon middle ear lesions of various kinds, but none so severe as in this case."

In Menière's first case the symptoms were *due to lesions of the internal ear.* The disorder may supervene in various ways quite suddenly, as in the case of Menière, or in that of the apprentice boy, already cited; or very gradually occurring in connection with chronic disease. The condition may also be fatal, or it may pass away quickly, or it may last for a long time without endangering life.

With regard to the treatment of Menière's disease, you must seek to determine the particular form of the malady with which you have to deal. If the cause be situated in the external meatus, try to remove it. If it be due to obstruction of the Eustachian tubes, seek to have them opened up. If it be due to changes in the labyrinth itself, much less can be done; but by means of counter-irritants and absorbents good results may be obtained. Charcot has spoken strongly of the advantage following the use of quinine in this disease, and remarks that it often seems to relieve the patient from the humming or ringing sounds which are so often complained of.

Lesions of the spinal cord are not very often attended by giddiness, but in cases of locomotor ataxia it is sometimes much complained of, and I have found it more prevalent in earlier stages of that disease than in the later. It may arise in various ways. Let me describe to you some cases. A very intelligent schoolmaster, resident in a country district in Cumberland, consulted me on account of spinal symptoms. I found that he had for several years had slight symptoms of locomotor ataxia; but that in consequence of overwork preparatory to inspection of his school, a degree of actual paralysis had supervened. This paralysis happily passed off in a few

weeks under treatment, but the ataxic symptoms persisted. He told me that he very frequently suffered from vertigo, but, on inquiry, it proved to have been present only when he also had double vision or actual squinting. I show you to-day also a patient suffering from locomotor ataxia. You observe that the gait is distinctly characteristic, and that, when his eyes are closed, he tends to fall. He complains much of giddiness, but this giddiness is cured when he closes one eye. It is a result of the double vision. In these cases then the cause is manifestly ocular, and referable to one of the groups already discussed. But that is not the only cause of giddiness in locomotor ataxia. It seems to result sometimes from changes in the sensory impressions, from other sources, and sometimes perhaps from changes in the brain.

Patients suffering from multiple sclerosis of the brain and cord often complain of giddiness. It may possibly result from the nystagmus, but certainly cannot be referred in all cases to this cause, for it may occur without nystagmus, and be awanting when nystagmus is present. It is apt to occur in short isolated attacks, and apart from any circumstance which seems obviously fitted to explain it.

Giddiness arises from a great variety of intra-cranial conditions, conditions varying both in respect of seat and of lesion. It occurs from lesions of the cerebellum, the crura cerebelli, the pons, the cerebral peduncles, the corpora quadragemina, and other parts of the cerebrum, and it arises from anemia, congestion, hæmorrhages, softenings, and new formations. At the same time it must be admitted that cases may be found on record of lesion in any one of the above-named intra-cranial structures in which no giddiness occurred, and it is equally true that each of the lesions may be unattended by the symptom.

Sometimes the giddiness appears to be a direct symptom; sometimes it merely results from paralysis, especially paralysis of some of the ocular muscles, attended by double vision. It is certainly most constantly associated with cerebellar disease. The peculiar swaying or staggering gait, like that of slight intoxication, has long been recognized as a feature of cerebellar maladies, and although the staggering is not always, it is very often accompanied by a feeling of giddiness. It is said to be generally increased when the patient opens his eyes, and

relieved when he lays hold of anything. It may be present only occasionally, or when the patient is in the upright posture, but sometimes it is present even while he is lying down. As a good typical case of cerebellar disease, I select one which is recorded by Professor Nothnagel as having been observed by himself. W. P., a servant girl, thirty-six years of age, had complained since the beginning of August, 1876, of a frequently recurring feeling of giddiness. It used to come on suddenly, especially when she was walking, or when she rose from her chair, and usually disappeared in a few seconds or minutes. At the same time she used to sway to both sides, but never fell to the ground. From the beginning of September, she had severe pain in the back of the head; from the middle of September she had frequently malaise and vomiting. Later on, she had frequently humming in both ears, and she observed an unpleasant saline taste in mouth and increased thirst. In the beginning of October, Nothnagel examined her, and found that intelligence was normal, that there was almost constant occipital pain, that she always lay on her back, because when she sat up she had giddiness and vomiting. The movements of arms and hands were free and strong. The finer movements were easily performed, even when the eyes were closed. There was no abnormality of the muscles cf the face, or the tongue. All movements of the limbs were rapid and precise, even with the eyes closed, and the patient knew the position of her limbs, and could point to them. She could walk if she got the slightest hold of anything; but if she were left alone, marked swaying occurred (in no particular direction), with giddiness, choking, vomiting, and the patient tumbled down if not held up. It made no difference whether the eyes were closed or open; without help she could not walk, and even with support she walked unsteadily. When giddy, the patient felt as if objects moved round her. The special senses were normal; the ophthalmoscope showed in the right eye slight venous congestion; in the left, a normal fundus. During October and November her condition remained unchanged; from the end of December she frequently had epileptiform convulsions and double vision. She died 18th January, 1877, within six months of the commencement of the illness. On the post-mortem examination, the cerebrum was found to be normal. The cerebellum was found to contain several tumors, involving

both sides and also the central part. The crura cerebelli and the corpora quadragemina were unaffected. In this case, you observe that giddiness was very pronounced, and that it preceded the occurrence of the other symptoms, was not due to squinting, and most likely resulted from the cerebellar disease.

As an instance of cerebellar apoplexy, I shall mention one observed by Dr. Brodribb of Hastings, and briefly recorded in Dr. Wilks' work on the "Diseases of the Nervous System." A lady, aged fifty-three, had travelled from London to visit a sick relative, and being depressed in spirits, had recourse, more than once, to her brandy bottle. After her arrival at the house, she went out for a drive, and on her return she could scarcely walk or stand. She expressed a fear that she was intoxicated, having partaken too largely of brandy, and her friends were at first willing to coincide in this opinion. After a few hours, however, she fell into a state of coma, and died. The post-mortem examination showed a clot of blood of the size of a pigeon's egg in the right lobe of the cerebellum. Dr. Brodribb, on further inquiry, could not learn that there had been any special loss of motion, or any disturbance of the senses, but merely a want of regulating movement, such as is seen in an intoxicated person.

With regard to the cerebellar reeling, you must observe that it is not always associated with giddiness. I lately had the opportunity of studying closely a case which I believe to be certainly cerebellar. A student, nineteen years of age, and who had been remarkably strong, and, as I was told, a leading athlete in his university, sustained an injury to his head about four years ago. At first this injury was followed by no symptoms, but soon there occurred squinting, with double vision and giddiness. This giddiness, he found, as I have already told you, that he could cure by closing one eye, and it disappeared permanently as the squint passed away, in the course of three or four months. From this time he remained free from any unhealthy symptoms, able to pursue his studies and his athletics until about six months before he came under my notice. He then observed that in walking he occasionally reeled to one side; and people remarked upon the peculiar gait, ascribing it to intoxication. But, while he thus staggered, he felt no giddiness. He noticed that the staggering was more toward the right side, and that his head tended to go in that

direction, and forwards; and he felt just as if he were making the extraordinary movement in order to maintain his equilibrium. He became unable to ride because of the tendency of his head to fall forward, a tendency which he could better overcome when on foot than when on horseback. Four months before I saw him, although other people constantly remarked upon his peculiar gait, he was able to go about pretty well; indeed, he walked a distance of six miles in deep snow, and appeared to be little the worse of it, so that his muscular vigor was evidently then little impaired. But for a marked tendency to constipation, I found all his organs and functions healthy, except the nervous system. He complained of a feeling of numbness in the limbs after exertion, but sensibility was unimpaired; his sight was good, and the fundus of the eyes normal. But he said that after walking or other fatigue his sight became indistinct. Hearing, taste, and smell were natural. With regard to his muscular sense it was difficult to speak; there was no definite proof of it being impaired, unless it be a proof that he staggered more when his eyes were closed. His skin reflexes were natural, the patellar tendon reflex slightly exaggerated; the organic, not interfered with. His voluntary movements were imperfect in consequence of a tendency to lurch to the right side and his head to pitch forward. He grasped firmly with the hand, but performed finer movements very imperfectly; in particular, could not write well, especially when tired, or when he had been attempting to study. I think that it can scarcely be doubted that in this patient cerebellar disease has occurred, probably as a consequence of the injury sustained four years ago. The gait is quite characteristic, and differs markedly from that of locomotor ataxia. I satisfied myself that he felt no giddiness even when the staggering was pronounced.

Two very interesting cases are recorded by Dr. Fraser, of Paisley, in both of which apparently an arrest of development of the cerebellum had taken place. In one, which proved fatal, the brain and cord were carefully examined, and it was found that the cortical grey substance of the cerebellum was little more than half its normal thickness, and that there were gaps at the surface, probably caused by collections of fluid in the membranes; that the white substance was less reduced in bulk, indeed appeared excessive in proportion to the grey; and that the cells of Purkinjé in the cortex were greatly shrunken and

contracted, their processes being indefinite and altered in direction.

The patient had been a healthy child till his second or third year, after which the disease had gradually developed. The first symptom was an occasional slight reel or stagger in his gait. About the age of six or seven this reeling became more marked, and almost constant. He was first seen by Dr. Fraser when about twenty years of age. At that time he was employed delivering newspapers. He walked with the gait of a very drunken man, reeling greatly, his body inclined forward, his head thrown back, apparently in constant danger of falling on his face. He was obliged to give up his occupation when about twenty-seven years of age, and was afterwards often seen running forward, then catching hold of a lamp-post, and then again going on. Although he constantly swayed about, there is no mention of giddiness in this case.

In the sister of this patient similar symptoms existed. She was unable to write in consequence of the want of power to make the necessary movements. She could not walk for fear of falling forward. Her disease had slowly advanced as the brother's did. She also was not usually affected with giddiness, indeed it was seldom a prominent symptom, except when she lay down. She then became giddy, or her sight grew indistinct. If she turned her head the feeling became exaggerated, and often she had visceral sensations, such as a feeling of nausea, but she did not vomit. It is certainly reasonable to conclude that in the female patient the lesion was the same as existed in the brother's case.

Here, then, under certain conditions, severe vertigo occurred, while usually it was not present, and apparently it was not the cause of her reeling.

Ferrier states that division of the cerebellum down the middle line, and lesions which are symmetrical on both sides, do not cause more than slight disturbance of equilibriation. Professor Schiff once showed me in Geneva two dogs on which he had performed cerebellar section. In the one, the gait was very staggering; in the other, the staggering was slight; and he told me that in the former he had injured one lateral lobe, in the latter the two lobes equally.

Ferrier has shown that where the anterior part of the middle lobe is injured, the animal tends to fall forward on his face. When the posterior part of the median lobe is injured, the

head is drawn backwards, and there is a constant tendency to fall in that direction. Ferrier satisfied himself on these points by experiments on a monkey, and clinical observations have confirmed his experimental results.

In lesions of the crura cerebelli giddiness often occurs, and the patient shows a tendency to fall towards one particular side. In illustration of this, I may cite a case recorded by Friedberg about twenty years ago, and quoted by Nothnagel in his admirable work on the Localisation of Brain Disease. A patient of six-and-twenty was, on 5th October, 1855, struck on the forehead, fell backwards upon the pavement, but was able to walk home. Fracture, with depression of the right parietal bone was found. On 15th October, symptoms of meningitis set in; the skull was trephined four weeks afterwards; the patient began to go out, and was quite well and able to work until August, 1856. Then he began to have occasional headaches, and they gradually became more severe. In October, attacks of giddiness, often attended by vomiting, came on. On the morning of the 24th of that month when he got up and was attempting to walk to the washhand-stand, violent giddiness occurred, and he was turned irresistibly to the left side, so that he could only make his way to the stand moving tumultuously, and in a half circle concave to the right. Next night he was again seized with intolerable giddiness, and suddenly began to turn round in bed on his long axis, from left to right. This was repeated five times. In the course of the same day three similar attacks occurred; first the head was turned to the right, then the thorax, then the under part of the body. When the body had reached the right side, the rotation went steadily on, the extremities sometimes followed voluntarily, sometimes seemed to strive against the movement. The eyes wandered anxiously here and there, seeking help. A complete attack lasted only a couple of minutes, as it then subsided. The patient continued to have frequent attacks of giddiness, and the feeling as if he were about to fall to the right, and as if it were necessary to support himself. On 21st November, he took a feverish turn, and his right leg and arm became paralysed. On 30th November he died. The autopsy showed the cerebrum natural, the left half of the tentorium cerebelli prominent. Under the left lobe of the cerebellum there was a rather fresh blood clot of about fifteen grammes. There was fracture of the right part of the occipi-

tal bone. There was exudative meningitis round the left lobe of the cerebellum, with red softening of its cortical substance; the inflammation of the pia mater extended along the left cerebellar peduncle to the pons, close down to that structure. The tissue of the peduncle was in a state of red softening, with small extravasations to a depth of from one and a-half to two lines; the other parts of the cerebellum, pons, and medulla oblongata were natural. In this case it is reasonable to assume that the giddiness resulted partly from the cerebellar, but probably also from the peduncular disease; the peculiar movements were characteristic of the lesion in the peduncles.

I shall cite another case which has been quoted by many writers, and was originally recorded by Belhomme. A lady of sixty had long suffered from giddiness and weakness of the legs, but was seized one day with an attack which lasted half-an-hour, in the course of which she was constantly constrained to turn round. A fresh attack followed upon great mental excitement. This time the tendency was to turn to the right. The attacks became more frequent, till at last they occurred four or five times in a day. The condition became gradually worse; the attacks assumed a pretty definite character: the patient suddenly lost consciousness, and drew herself together spasmodically; she turned herself with astonishing rapidity, usually to the right, but sometimes to the left. She died of bronchitis nearly ten years after the commencement of the illness. On examination, it was found that the middle cerebellar peduncles both showed a depression, the left more marked than the right, due to growth connected with the bone. On section through the pons, there appeared between the middle and posterior thirds a varicose injection of crescentic shape, the pons itself was one-third smaller than natural, the walls of the fourth ventricle softened and disorganised. In this case you observe that the symptom of giddiness had existed for more than ten years, and considering the character of the changes met with after death, it is reasonable to suppose that it was due to the pressure upon the cerebellar peduncles, although it may be maintained that they were referable to the cerebellum itself.

Giddiness is also observed in some cases of disease of the Pons Varolii, but I must defer illustrating this fact until the next lecture.

LECTURE III.

GENTLEMEN: At the end of last lecture I was about to speak of giddiness as a symptom in cases of disease of the Pons Varolii. But before doing so I wish to show you a typical case of cerebellar disease, which I am able to show you by the kindness of Professor Fraser.

You observe that, even as he stands, this patient sways about in an irregular manner. Now, when he attempts to walk he staggers like a person in a state of intoxication, and even when I make him sit down, you perceive that he seems inclined to fall in one direction or another, and then to catch himself by a sudden effort. He tells me that he constantly feels giddy, but I find that his giddiness is increased by his movements. If the back of his head is pressed against the wall, or if it is held firmly, so that the jerking is prevented, his giddiness almost, but not altogether, disappears. It is thus, then, in some degree, a consequence of the movements, but it is not entirely dependent upon them. It is due in part to some sensory change.

And now, to return to the Pons Varolii,—I shall cite in illustration one or two cases. Guéniot describes the case of a woman forty-seven years of age, who was suddenly seized with giddiness, with loss of power of speech, and paralysis of the left arm and leg. These conditions persisted. The tongue was paralysed, but there was no difficulty in swallowing, intelligence was good, and there was no affection of the facial muscles or those of the eye. Sensibility was retained in the paralysed parts—apparently rather increased; death followed in four days. On examination, it was found that the brain was natural, but the pons broken up by clot. On closer examination of the specimen it was found that while the right pyramidal bundle was completely destroyed, the left was scarcely affected. The point of interest for us in this case is the giddiness. It was manifestly a prominent feature, and it

was not the result of paralysis of any of the muscles of the eye. No cause was found excepting the lesion of the pons, and to it the well-marked vertigo must manifestly be ascribed.

Another case may be cited, which was observed and recorded by Professor Nothnagel. A syphilitic man of forty-six was seized with headache and giddiness, with tendency to fall to the left side, and loss of consciousness. After two or three attacks of that kind, he was admitted to hospital, a permanent condition having become established; there was no paralysis of face, eye, or tongue, and speech was normal; but there was complete motor paralysis of left arm, and parasis of left leg. Sensibility was unaffected. The reflexes could not be produced in the left arm. For two months there was no change, and in particular no tendency to muscular contracture. Death resulted from pneumonia; and on examination, it was found that a mass of softening, of the size of a hazel nut, was situated on the upper half of the right side of the pons, near the cerebral peduncle. In this case, also, you observe that giddiness was a prominent symptom, and that softening was the only change found on examination.

It being clear, then, that lesions of the pons itself are capable of producing giddiness, even when double vision is absent, it is scarcely necessary to adduce proof of it resulting in cases where squinting occurs. But such cases have also been observed.

In lesions of the cerebral peduncles giddiness is sometimes seen, but usually it results from squinting due to paralysis of some of the muscles of the eye. Such a case as that which I am about to state, suggests that other influences operate here. It is recorded by Rosenthal and quoted by Nothnagel. A woman of thirty-nine had suffered for two years from headache, giddiness, and dimness of sight, with paralysis of the right side. The giddiness was so great that the patient preferred the recumbent position. Severe giddiness set in if, even when recumbent, she turned on her left side. She had paralysis of the third left cranial nerve with paralysis of the lower branches of the facial and of the limbs; on the right, distinct anæsthesia. After death, it was found that a tumor of the size of a pea lay between the crura cerebri. The innermost part of the right cerebral peduncle was softened. In the interior of the left crus cerebri there was a cist of the size of a

pea, which destroyed the left third nerve, and another tumor of the size of a bean involved the right oculo motor.

I think you will agree with me in believing that although the squinting may have been a factor in the production of giddiness, there certainly was a further influence in operation, seeing that the symptom was so severe.

In lesions of the corpora quadrigemina giddiness sometimes occurs, at all events staggering gait has been observed in some cases. In illustration of giddiness from this lesion, I select a case recorded by my friend Dr. Duffin, of King's College, London. The patient was a steady and powerful man, and the earliest symptom—a sense of dragging at the back of the neck—occurred about four months before his death. The walk early became unsteady, and there were repeated attacks of vertigo which diminished when the eyes were closed. Headache and contraction of the muscles of the neck and spine were prominent symptoms. Sight soon became impaired, and blindness speedily followed with well-marked double optic neuritis. The intelligence became obscured only about a fortnight before death. He died comatose. The autopsy showed the corpora quadrigemina and the pineal body replaced by a glioma. The right optic thalamus had atrophied from pressure, and the right superior peduncle of the cerebellum was also involved. It is possible that the giddiness was connected with change in the peduncle, but considerng that the tumor involved primarily the corpora quadrigemina, and the pineal gland, and that the giddiness was an early symptom, it is more reasonable to refer the giddiness to the altered condition of the corpora quadrigemina themselves.

Besides these distinctly localised intra-cranial causes of giddiness, there are others which seem to act upon the cranial contents generally,—for example, cerebral tumor in any part of the brain may be found to produce occasional fits of giddiness, and alterations of the cerebral circulation induce a like result.

Congestion, whether of active or passive origin, induces giddiness, sometimes to such an extent as to prevent the patient from walking, and often sufficing to render quick turning round a matter of difficulty. In treating such a condition, one must first advert to the cause. If it be passive congestion, seek to remove the obstruction; if it be the result of

plethora, diminish the volume of the blood; if it be from overwork, or other undue excitement of the brain, or from a tendency to inflammation, use such remedies as cold to the head, ergotin, the bromides, and perhaps derivatives.

Vertigo is a common symptom in cerebral anæmia. It is apt to be induced when the patient suddenly changes his attitude, either by lying down or getting up. Sometimes it is merely what the patients describe as swimming in the head, at others it is well-defined vertigo. It is often associated with neuralgic and other forms of headache. You will meet with it very frequently in cases of chlorosis. Indeed it is sometimes one of the most troublesome symptoms of that disease. It is also occasionally met with in cases of anæmia due to loss of blood, slight and long continued, as in bleeding hæmorrhoids, or more formidable as in menorrhagia or other uterine complaints. Sometimes, also, it is seen in the later stages of aortic valvular disease, when the cerebral anæmia becomes pronounced; and sometimes in cases of unexplained anæmia whether referable to the pernicious variety or not, it also occurs. In these cases you will find that the patient suffers less (except when special peculiarities come into play) after food has been taken, or after a little alcohol, and that he is sometimes markedly relieved, for a time at least, by the use of nitrite of amyl. The treatment of the giddiness in such cases is, of course, the treatment proper to the variety of anæmia which may be causing it, and usually consists in checking any drain of blood which may exist, and improving the condition of the blood itself. Let me remark in regard to one of these causes—viz., the bleeding from hæmorrhoids,—that you will often have to deal as physicians with this complaint, and that I know of no plan of treatment more successful than that which I learned from Professor Oppoltzer, of Vienna—viz., the regular use of aloes and sulphate of iron. If you will direct your patient to take morning and evening, or three times a-day if it be necessary, a pill containing one grain of aqueous extract of aloes, one-third of a grain of sulphate of iron, and two grains of extract of taraxacum, you will find that free soft motions are produced, while the bleeding as a rule rapidly disappears. In this way your patients may often be relieved without having recourse to surgical treatment. I say nothing of the treatment of the uterine or other hæmor-

rhages; but should like to accentuate the fact that in the effort to improve the condition of the blood, when our chief remedy, iron, fails, arsenic often stands us in good stead.

Vertigo is sometimes due to mere exhaustion of the nervous system, as from overwork, sexual excesses, and such like; this occurs quite apart from anæmia, is manifestly a result of changes in the nerve centres themselves, and is to be treated by means of rest, good diet, and nervine tonics—chalybeates often proving unsuitable.

Giddiness sometimes results from and persists for long after injuries to the head. The patient is thrown from horseback or from a bicycle, and, falling on the head, is for a time stunned; on recovering, he is at first much troubled with giddiness or with headache, and he finds that for months, or for life, he is much more easily made giddy, becoming so under conditions in which previously no giddiness would have occurred. The nervous system has acquired a new mobility of this and of other kinds. You will find that patients who have suffered from sunstroke, even to a slight extent, often exhibit the same peculiarity.

A very distressing form of vertigo is that which is associated with epilepsy. One often meets with such cases in practice. Take as illustration a case regarding which I was recently consulted. A lady, aged forty, sustained an injury to the head about seven years ago. From that time she became subject to attacks, usually merely of giddiness, but occasionally of complete unconsciousness. On examination, nothing was found in the state of the ear or of the alimentary tract to account for the condition, and the only satisfactory explanation was that which referred the process to the category of epilepsy. Such a diagnosis as this can scarcely, of course, be established in the absence of more typical epileptic attacks. The treatment in such a case must consist of careful regulation of diet, the administration of bromides, and belladonna, and, in rare cases, operative interference at the seat of injury in the skull. You must also note that the epileptic may have giddiness as a warning at the onset of his attack, sometimes as an aura, sometimes merely as one of the early phenomena of the attack.

In other nervous affections, such as migraine, giddiness occurs. It is not in my experience a frequent symptom, but

is met with occasionally towards the end of the first stage, when the ocular or other sensory troubles are nearly passed, and the headache is about to commence. Sometimes it occurs later, when the headache is fairly developed, and the nausea or vomiting has come on. The diagnosis of the cause of the form of vertigo is easy to any one who is acquainted with the features of migraine. The treatment in such a case is of course that applicable to migraine generally; and that again is guided by a study of the cause, whether it be connected with gastric and hepatic derangements, with constipation, or with nervous exhaustion. The mention of each of these causes will suggest to our minds the appropriate remedies; but, in addition, you will find that, in many cases, the attacks may be warded off by the regular use of the bromides, or of guarana, or chloride of ammonium, or of an alcoholic stimulant, or hyoscyamus, or spirit of chloroform, or even a cup of tea or coffee.

Giddiness is often complained of by old people, particularly those affected with degenerative changes in the arteries, and in such cases is a cause of much alarm, being popularly and justly associated with the idea of a tendency to apoplexy. Whatever may be the precise pathology of this variety, I would have you bear in mind that one very able physician, Dr. Handfield Jones, has found the bichloride of mercury, in small doses, of very great service in such cases.

There is a form of intra-cranial giddiness which you can easily produce experimentally for yourselves,—viz., that described by the late Professor Purkinjé of Prague, as being produced when a constant current of sufficient strength is transmitted through the head from ear to ear. If you place the electrodes in the mastoid fossæ, and allow a current of medium strength, say ten or twelve cells, to pass, you will find immediately that giddiness results, external objects, appearing to rotate in the direction of the current, while you incline to turn in the opposite direction. It requires a much stronger current to bring out movements of the eyeballs, such as Hitzig has described. It may turn out that these changes are due to an influence exerted on the semi-circular canals; but this seems improbable, both from the locality in which the electrodes have to be placed, and from the occurrence of the nystagmus when stronger currents are employed.

With regard to the diagnosis in cases of giddiness from intra-cranial disease, I should advise you to satisfy yourselves first as to whether there is paralysis of any of the eye muscles or not, and not to conclude that giddiness is due to brain lesion unless other and more characteristic symptoms of intra-cranial disease be present.

The treatment of giddiness from intra-cranial causes, other than those already referred to, is not very promising at present. There are some cases in which the lesion being syphilitic, iodide of potassium, or mercury, or a combination of the two, are of great service; there are probably some in which the bichloride of mercury is useful, even when the disease is not due to syphilis; but on the whole, medicine is of little service. Now and then a surgical operation does good by removing some irritating fragment of bone, or by diminishing intra-cranial tension.

Having thus traced the various nervous causes of vertigo with which we were acquainted, I now proceed to describe a number of others which you will find come frequently into operation. Among those we shall take first dyspeptic giddiness. It is matter of common observation that, along with gastric and hepatic disturbance, headache and giddiness frequently occur. But there is in addition a formidable and alarming kind of giddiness, which has been well described by Trousseau in his clinical lectures, and by Dr. Ramskill in "Reynolds' System of Medicine." It is known by the name of stomachal vertigo, or *vertigo a stomacho læso.* The case generally presents such features as the following: The patient an hour or two after taking food is subjected to some fatigue, excitement, or emotion, and suddenly becomes very giddy. He reels, perhaps falls to the ground, becomes sick, may lose consciousness, or may simply suffer from a peculiar headache. He seems to himself to be turning over in all directions, or to have currents within his head, which are flowing, in a curved direction, from before backwards, or from behind forwards, or in an irregular manner. Surrounding objects seem also changed. The ground feels to his feet like the deck of a ship on a stormy sea, while other things seem to be constantly changing their position, swinging with jerks upwards or downwards, backwards or forwards; and while he knows that these are illusions, he yet cannot steady himself. Per-

haps vomiting takes place; perhaps there is a copious discharge of flatus from the stomach or the bowels; perhaps sudden diarrhœa. Then he becomes almost well, the giddiness passing off, and he is free from the illness for the time. Now, it may be that you find—as Dr. Ramskill found in one of his cases—that the patient has had a hearty breakfast, including sausages and Devonshire cream, or some other things equally difficult of digestion, or the diet may have been perfectly simple. One attack seems to render the patient more liable to the recurrence, and your patient may give you the history of many seizures of varying severity.

This form of vertigo may be transient, or may be persistent for a time. Some years ago I was consulted by a lady whose home was in a distant part of Scotland. She was little and stout, and came into the room with a curious dazed expression, puting her hand on a chair or a table at every opportunity for support, and still occasionally reeling a little like a person on board ship who has not acquired his sea-legs. She stated that life was made intolerable to her by various uneasy feelings, the billowy rolling of the floor being the most distressing; but that occasionally she had fits of unconsciousness, and sometimes convulsions. Her tongue was furred; her appetite was impaired; and she suffered from constipation. There was no deafness nor noises in the ears, nor any other condition characteristic of Menière's disease. Neither was there any fixed pain in the head, nor eye changes, paralysis, or convulsions, such as might have been indicated by cerebral tumor, and her illness had already lasted for years. I was satisfied from the result of treatment, as well as afterwards from the absence of important symptoms, that this case was to a large extent dyspeptic in its origin, the dyspepsia being implanted upon a very sensitive system. The fits of unconsciousness I regarded as syncopal, the convulsions as probably hysterical. When she was treated by means of stomachics and aperients, with regulation of the diet, she became greatly better, and now enjoys fairly good health.

A patient lately presented himself to me much alarmed about attacks of vertigo. He was somewhat advanced in years, of large frame, and had retired from professional work, which had, he told me, been laborious. As he suffered much from feelings of debility, he took food at short intervals and

in considerable quantity; being unfit for active bodily exertion, he tended to grow stout. Gradually he became subject to attacks of giddiness, associated with extreme anxiety, dimness of vision, and nausea, and even vomiting. The vomiting was attended by partial relief, and was followed after an hour or two by complete recovery. In this case also there was nothing characteristic of labyrinthine or cerebral disease, and I confidently anticipate, that with the establishment of better habits, as to diet and regimen, and with the regular use of stomachic and aperient remedies, he will quite recover.

In the treatment of dyspeptic vertigo, Trousseau had the utmost confidence in the use of alkalies and bitters; he used to give a dose of quassia every morning and at bedtime, and after meals a powder containing 5 grains of bicarbonate of soda and 5 grains of magnesia with 10 grains of prepared chalk; the powders were occasionally alternated with alkaline mineral waters, but a not unimportant part of the treatment was the insistence on careful dieting and regular bodily exercise.

Remember, then, that as to diagnosis, the hints that I have given you will guide you, and that in the treatment of this form of giddiness you will often effect a cure, and constantly obtain great relief for your patient by very simple means. Regulate the diet as to quality, quantity, and times of eating. Give aperients if they are required, alkalies, and bitters. Give stomachic remedies, such as rhubarb, bismuth, soda, and aromatic powder, followed by nux vomica, quinine, or calumba or chiretta, and the good effects will speedily manifest themselves. When there is much flatus in the stomach, carminatives often do good, and if the ordinary ones fail you will find that from ten to fifteen drops of turpentine may give great relief.

Now, what is the nature of this form of vertigo? Is it reflex? Is it a result of alterations of circulation within the brain? Is it a result of poisoning? It is impossible, in the present state of our knowledge, to answer these questions definitely, but I lean to the opinion that it is of the nature of poisoning, that in connection with the chemical changes of the digestive process under certain conditions, some substance is developed which, like alcohol or tobacco, acts upon the nerve centres so as to induce the peculiar uneasiness.

Among the commonest form of vertigo must be reckoned

the toxic, and among them those due to alcohol and tobacco. When the action of alcohol has reached a certain point giddiness is experienced, and associated with this is the reeling gait. Objects may appear to whirl round the patient, or he may appear to be rotating—the movement may seem to be irregular, or in certain definite directions, as from right to left, or from left to right. You know the case of a well-known legal luminary in bygone days, who was found by a friend standing rather unsteadily in the square in which he resided, making no progress towards his door. When interrogated as to his object, "Don't you see," said he, "that the square is going round and round? and whenever my door passes I'm going to jump in." Or the apparent movement may be a rotation on a horizontal axis, as in the case of the man who was found lying on the ground, and who explained that the earth was turning rapidly round, and he was holding on by the grass to prevent it. We may not be in a position at present to state positively how this giddiness is brought about, whether through alterations of the vascular system, or by direct influence upon nerve centres, but the latter seems to me the more probable explanation. The effect of tobacco on those unaccustomed to its use is similar to that of alcohol in the production of giddiness; and other toxic agents might be mentioned which induce a like result—such as stramonium, belladonna, hydrocyanic acid, lobelia, hyoscyamus, and veratrum viride.

Occupying an intermediate position between the toxic and the dyspeptic, and perhaps also related in some measure to the labyrinthine vertigo, is the variety occasionally met with in gouty patients, sometimes slight, sometimes really severe, and which is apt to occur in the intervals between regular attacks of gout, especially as the attacks draw near, and which is often absent for a considerable time after the typical seizure. This form is, in my opinion, often referable to the stomach, but it may also perhaps be induced by toxic influence upon the nerve centres.

Besides these obviously toxic forms of vertigo, there are others which are reasonably to be referred to the same category—I mean the giddiness occurring in acute specific diseases. In the commencement of fevers, the symptom is not uncommon—just as we have commonly rigors and headache, and

occasionally convulsions, as initial phenomena. It has been remarked that in influenza it is frequently very troublesome; —sometimes as one of the earliest indications of morbid action; sometimes during the height of the disease; and that occasionally it continues for long after the acute stage of the malady has passed. It bears the same relationship to many acute diseases. In some of these cases, it is probable that at first a direct toxic influence is exerted, while at a later stage exhaustion of the nerve centres may be regarded as the most likely cause. In the case of influenza, it may be reasonably assumed that catarrh of the Eustachian tubes accounts for it. In the way of treatment you will do well to keep the patient at rest and in the recumbent position, as free as may be from the exciting causes of giddiness; for you will find that sitting up or attempting to read will often suffice to induce an attack. During the later stages, tonics and nutrients, with moderate doses of alcohol, prove most serviceable.

Vertigo is sometimes produced by violent fits of coughing; sometimes by violent fits of sneezing. I do not know for certain the explanation of the fact; it may be due to changes in the ear, possibly to changes within the sensorium. It is not of very frequent occurrence. I think it is most likely due to the first-mentioned cause operating through the Eustachian tubes. The correctness of this view could be easily tested if one were to meet with a patient subject to the symptom who at the same time had obstruction of these passages.

In some cases giddiness is due to peripheral irritation, and that irritation may be situated in various parts of the body. As a typical example, I select the giddiness which sometimes results from the presence of worms within the alimentary system. You are aware that other nervous symptoms, such as convulsions and cough, arise from this cause, and we may assume that the vertigo is brought about in the same way as these other symptoms. It is most reasonable to refer them, in the present state of our knowledge, to reflex action, and in the case of giddiness, to an influence upon the cerebral circulation. The treatment in such a case is, of course, obvious. Let the irritating cause be removed from the intestine, and the giddiness will disappear.

Vertigo arises in some individuals from purely mental causes. A patient goes on board a steamer, and although there is no

tossing, and no perceptible vibration, the recollection of what he has suffered before, or the sight of the oscillation of the waves, makes his head swim, or renders him actually giddy. In some, giddiness supervenes if a child is seen whirling rapidly round. I have known it even produced by another person walking in a small circle. The very thought of giddiness suffices in some to induce the sensation.

We have thus passed in review the chief causes of this sensation. We have considered giddiness as arising:—

(*a*) From external conditions.
(*b*) From changes in the sensory structures.
(*c*) From changes in the conducting fibres.
(*d*) From changes in the central nervous system.
(*e*) From dyspepsia.
(*f*) From toxic causes.
(*g*) From causes of uncertain seat.
(*h*) From peripheral irritation.
(*i*) From mental causes.

By following out the indications which I have given, you will generally be able to determine with certainty the particular cause in any individual case, and thereby be able to devise a rational treatment.

A CRITICAL STUDY

OF THE

Clinical Value of Albuminuria in

BRIGHT'S DISEASE.

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PREFACE.

DURING my last year of service as interne under Professor Dieulafoy, I often had occasion to study renal affections, and I was frequently surprised to find that statements which had been accepted as classical facts were in reality not such at all. We have thought it might be useful to collect our experiences in a single monograph in order to fix, after a definite fashion, the question of the connection of albuminuria with Bright's disease, including under this term, as does my teacher, all the chronic inflammations of the kidneys.

What clinical value should we to-day accord to the symptom albuminuria in Bright's disease? Such is the question we have endeavored to answer, and to which we will try to reply at the end of our critical study.

We have opened our study by a chapter devoted to chemistry, for it has seemed essential to precede our clinical study of albuminuria by a chemical study of albuminous urines. In this first portion of our work we have sketched quite completely the methods of examining the urine. In this work we have never forgotten that we should not be chemists but only clinicians occupied with chemistry.

We have next taken up the clinical study of the symptom

albuminuria in Bright's disease, and this portion we have divided into two distinct parts of unequal importance.

In the first part we have noted, at the outset, the fact that albuminuria has lost its old diagnostic value; next we have aimed to find out its actual value, taking successively into consideration its existence, nature, and its relation to the urinary secretion and to the general state of the patient.

We have finally reached the conclusion that albuminuria, while it is alone of limited value as a symptom, is, when associated with the other symptoms of Bright's disease, of decided importance.

In the second part we have endeavored to weigh the prognosis of albuminuria in case of Bright's disease. We have been obliged to admit that the presence of albumin in the urine is of slight importance, and that not it, but the toxicity, so to speak, of the urine should be the guide to our prognosis.

After relating experiments which have seemed to us of interest, we have stated in our last chapter the actual clinical value of the symptom albuminuria in Bright's disease.

VALUE OF ALBUMINURIA IN BRIGHT'S DISEASE.

CHAPTER I.

THE METHODS OF DETERMINING ALBUMIN IN THE URINE.

THE examination of the urine for albumin is to-day a rigid rule of practice, but the belief is current that the search requires few precautions, and that it is devoid of difficulties. Every day clinical errors are committed which are very prejudicial; every day the physician is mistaken by not discovering the albumin which exists, or by finding it where the urine does not contain it.

This is the reason why it seems essential, before entering upon the study of Bright's disease, to dwell somewhat at length on the precautions which should be taken in the determination of albumin in the urine.

SEPARATION OF THE MUCIN.

When we wish to test a specimen of urine for albumin it should first be filtered, in order to separate the mucus which is present even in normal urine (in small amount, true enough) and which is always found in large enough proportion in albuminous urine.

Usually the mucus is found in the form of a slight cloudiness, which settles little by little to the bottom of the bottle and which is readily recognized when looked for by transmitted light. At times, when the quantity of mucus is very large, the cloudiness becomes very marked, and, after an interval, a mu-

cous sediment, more or less abundant, forms, and after a little practice the quantity may be approximately determined. When the urine is filtered the mucus remains on the filter paper as a viscous mass having the appearance of varnish after desiccation. Nevertheless, after filtration there always remains a certain amount of mucous matter in solution which gives the chemical reaction of mucin. This should be at once eliminated, for in certain of its characteristics it resembles albuminoid matter, which might lead to error and cause us to think that albumin is present when it is not. In order to accomplish our purpose we have at our disposal the basic acetate of lead and acetic acid.

The basic acetate of lead, in solution, poured in small amount into urine, determines an abundant whitish precipitate which may be separated by filtration. The urine may be considered as containing no more mucin when, tested again by the lead solution, it gives no further precipitate. It is then perfectly clear, free from mucin, but it contains lead, which must be eliminated by the sulphide of hydrogen, which precipitates the lead under the form of black sulphide, which is separated by filtration.

This process, recommended by many writers, has many objections: in the first place, the basic acetate of lead, although it precipitates the mucin, does the same by many salts (the urates, the phosphates, the chlorides, etc.), so much so that one to two drops of this solution causes an abundant whitish precipitate. Further, in order that the precipitation may be complete, considerable time is required. Finally when, after filtration, we wish to eliminate the lead, the obstacles are many; the elimination, in order to be complete, requires at least two days. For these reasons we have given up this procedure, scarcely practical from a clinical point of view.

The acetic acid reaction remains. Although its value in eliminating is open to discussion, since it only precipitates incompletely, we do not hesitate to give it the preference; it is besides the only procedure recommended by chemists whose competence is not in dispute, such as Wurtz[1] and Neubauer and Vogel.[2]

When to a urine containing mucin we add acetic acid, a uniform cloudiness is obtained, which becomes much more visible if, following the advice of Lobisch,[3] we add in addition

to the acid, a small amount of iodine. This cloudiness only disappears on the addition of an excess of acid, never under the influence of heat (characteristics which differentiate from precipitates of uric acid or of acid urates, forming in excessively acid urines); it disappears, on the other hand, instantaneously and completely if at once a few drops of chlorhydric acid are added.

Only exceptionally and after long standing is a flocculent precipitate seen. Nevertheless if, before the addition of acetic acid, the urine has been diluted a number of times by its volume of water, there appear (when the quantity of mucin is not too small) quite large flocculi which under the microscope appear as quite fine granular masses containing a few crystals of uric acid. By proceeding thus the cloudiness due to urates is never obtained, and to this we will refer very shortly.

Verification of the Reaction of the Urine.

The urine having been made perfectly clear, and been entirely deprived of mucin, the reaction must be tested, and this is readily accomplished by means of litmus paper. The kind we recommend is that slightly tinted, a blue bordering on red; such a paper, prepared after Berthelot's method, besides being exceedingly sensitive, has the advantage of answering in all cases; it reddens distinctly in acid urine, and turns blue besides in alkaline urine; the result is instantaneous and the test is of the simplest possible kind.

The urine should be acid; it should redden distinctly the test paper, the color should be a frank red and not a reddish violet. Now frequently enough in cases of Bright's the urine is neutral or alkaline; if we do not wish to fall into error, therefore, it is necessary after having tested to acidify if need be by the addition of acid. Two acids have been recommended for this purpose: nitric and acetic acid.

The first has the great disadvantage of precipitating albumin if added in considerable quantity, and it renders void other reactions if added in great amount. This latter point is important to recognize, else we may fall into great error; we insist on this point, since many examiners appear to ignore it.

The fact, however, has been for long proved by Professor Jaccoud: "When you add," he says, "to albuminous urine a drop of nitric acid, this urine will not precipitate on being

heated;" and the same point has been noted by Bence Jones. This artificial and exaggerated acidity of the urine causes some modification or other of the albumin, which takes away its property of coagulation on boiling; and the proof is that if we add to the boiling clear urine a little dilute ammonia, in order to bring it back to its normal acidity, the latent albumin appears as a precipitate. This fact, little known, is most interesting; it proves that nothing can take the place of the test by litmus paper.[4]

Esbach[5] has latterly clearly proved the same point. By his directions, let us place in a graduated tube 10 cubic centimetres of a urine rich in albumin (containing, for example, 15 to 20 grammes to the litre) and then add two drops of nitric acid and heat; no cloudiness occurs.

This experiment, which we have over and again performed, must be emphasized, for it brings into strong relief the fact that a very small amount of nitric acid added to an albuminous urine completely prevents the coagulation of albumin by heat. Whatever the reason, whether it be due to the dissolution of the salts in combination with the albuminoids, as Albert Robin claims,[6] or not, the fact is well established, and it suffices to cause us to reject absolutely the use of nitric acid for the purpose of acidifying the urine of Bright's disease.

Acetic acid, on the other hand, does not precipitate albumin, and in nowise prevents its coagulation by the reagents ordinarily used; and therefore it is customarily used.

Certain precautions are still necessary in the use of this acid; it should be added only drop by drop, shaking the urine constantly, and we should cease adding when the reaction is clearly acid; an excess of acid stops completely the precipitation of the albumin. Acetic acid, we know, produces in most specimens of urine a uniform cloudiness which does not disappear on the addition of an excess of acid; this cloudiness, which may readily be mistaken for an albuminous precipitate (Reissner), is due to mucin; by acting as we have advised—that is to say, by clearing the urine of this substance—error is not likely.

Determination of the Albumin.

It is only after the two preliminary experiments which I have stated that we should proceed to search for albumin.

Many methods have been proposed; I will only mention the principal ones, dwelling particularly on those which are habitually resorted to, and these are heat and nitric acid.

Heat.—Under the influence of heat albumin coagulates; a large amount results in a flocculent precipitate, and a small amount produces a more or less marked cloudiness. The clear urine is poured into a tube half full; holding the tube by the base the upper part is alone heated; at 50° to 60° a distinct cloudiness is observed, the more readily recognized since the lower portion of the urine is clearer, and later on, at 75°, the albumin coagulates. (These two distinct phenomena possibly result from the successive coagulation of the globulin and the serin.)

After this first attempt the urine is entirely heated and allowed to settle; the albumin gathers in more or less numerous flocculi at the bottom of the tube.

The cloudiness or the precipitate thus obtained has the characteristic of not disappearing on the addition to the hot urine of acetic acid drop by drop. This verification should be resorted to in every instance, for if under these conditions (heat and moderate acidity) there results or there persists a cloudiness, however slight, the urine certainly contains albumin.

This simple process may, however, give rise to errors which it is well to bear in mind. Under the influence of heat there occasionally forms in non-albuminous urine a more or less accentuated cloudiness, sometimes even a precipitate. This latter is due to the presence of phosphates (usually the bi-basic phosphate of lime) retained in solution by free carbonic acid. This gas being expelled by the heat the phosphates precipitate in flocculi which at first sight it is difficult enough to distinguish from coagulated albumin. In case the verification just spoken of is here resorted to, as soon as a few drops of acetic or of dilute nitric acid are poured in the urine becomes clear, and concomitantly, if the urine be hot, bubbles of carbonic acid are given off.

In other instances, albuminous urines give no precipitate by heat; they only become cloudy or milky; yet when to the boiled urine nitric acid is added to strong acid reaction a precipitate forms.

This happens most frequently in case of alkaline or neutral

urines; the occurrence is exceptional in acid urines. This source of error is thus readily avoided if care is taken as we have advised to test the reaction of the urine.

Nitric Acid.—When nitric acid is added to an albuminous urine a precipitate is at once formed; in case the urine contains much albumin the precipitate is white and abundant; sometimes the urine becomes solidified; in these instances there can be no doubt as to the presence of albumin.

The same does not hold true when the urine contains but little albumin; in such a case a slight cloudiness may escape notice, or a cloudiness due to urates may simulate an albuminous precipitate; the urates, indeed, becoming decomposed by nitric acid, occasionally form a greater or less cloud; but this disappears on the addition of acetic acid and there results with ammonia the reddish-purple color which is characteristic of uric acid.

When nitric acid is used for the test, it is poured in slowly in order to allow the reaction to form; in order to obtain the maximum effect it is usually sufficient to use the acid in the proportion of 1 to 10 of the urine. If much more of the acid is used the albumin is almost at once transformed into xanthoproteic acid, the orange yellow color of which may readily veil the presence of the substance for which we are searching.

Furthermore, if we have reason to suppose that albumin is present in trifling amount it will be wise to resort to Gubler's [7] or to Heller's [8] method, or, better still, as we are personally in the habit, to the following method, which is a modification of Heller's; it is, in fact, a very delicate method and requires only a small amount of urine.

In a graduated tube pour 1 to 2 c. c. of nitric acid and then let glide, drop by drop, along the side of the tube the urine which we are testing. If this experiment is carefully performed the result is very perfect; we obtain, from above downwards:

A layer of acid, perfectly clear, colorless;

A colored line at the level of the junction of the urine and the acid;

At the same junction, if albumin be present, a whitish ring more or less pronounced is formed.

Below this is the urine, clear or cloudy from deposit of urates.

Heat and Nitric Acid.—Heat and nitric acid, such are

the agents daily in use in the search for albumin. Since each alone may lead to error, it is customary, in order to obtain a positive verdict as to whether albumin be present or not, to resort to each successively. This custom dates back almost to the discovery of the symptom which forms the object of our study.

Wells,[9] in fact, one of the first to study this interesting question (1798 to 1812), having determined over and again that the same urine tested successively by heat and by nitric acid did not always behave in the same way, advised that every specimen of urine should be subjected to the two tests. This advice, which has met with the approval of his successors, is very just; and to-day, therefore, it is a matter of routine.

Now that we are amply familiar with these two methods of use clinically, the worth of which is unquestionably very great, especially when they are associated, we deem it essential to sketch briefly the other methods which have been advised for the determination of albumin in the urine. They may be all ranged under one or another of the following:

The first aim at simplifying the *modus faciendi;*

The second are useful for determining traces of albumin which would not be revealed by heat and nitric acid.

Methods for Simplifying the Test for Albumin.

Although the methods in routine use are very simple and require neither much time nor complicated instruments nor reagents, it is an established fact that although they may readily be resorted to in hospital work they are ill adapted for private practice, at least *ex abrupto;* whence loss of time and the lack of a point essential for the completion or the affirmation of the diagnosis.

Professor Potain[10] recommends the heating of the urine in an iron or a silver spoon over a spirit lamp or the flame from cotton in a saucer of alcohol; then to instil into the urine a few drops of vinegar from the end of a match.

Less summary methods have latterly been recommended by Hindenlang, Hoffmann, and Oliver.

Hindenlang[11] recommends metaphosphoric acid, which can be much more readily carried than nitric acid; it may be obtained in the shape of crayons, which may be carried in a

glass tube. Before use the end of a crayon is to be dipped for a few seconds in 2 to 3 c. c. of water, and we thus obtain a solution of metaphosphoric acid, which at once precipitates albumin. The crayon need only be dried and replaced in the tube.

We have used this method on several occasions, have always obtained very satisfactory and very rapid effects, and we therefore recommend it in the belief that it will often render signal service.

An equally satisfactory method is that recommended at about the same time by Hoffmann [12] and Oliver; [13] it is a trifle more complicated than the preceding, but we can by means of it determine rapidly if a given specimen of urine is albuminous or not.

The reagents of use are prepared as follows:

Strips of paper are plunged into a solution of citric acid, and others into a bath of potassio-mercuric prepared as follows:

1.	Corrosive sublimate, . .	1 gramme
	Aquæ destil, ...	20 grammes

Solve

2.	Iod. potass, . . .	1 gramme
	Aquæ destil, ...	2 grammes

Solve.

Mix the two solutions and the result should be a clear fluid.

The strips of paper are to be dried after having been impregnated with these solutions.

To test the urine: verify its acidity; if it is not frankly acid, if it does not color test paper brick-red, acidify with a citric acid paper. This point must never be neglected, else the result will be erroneous. Next place the reactive paper in the urine, and if albumin is present (even in feeble proportion, 0.50 gramme to the litre) it is precipitated to the bottom of the test tube.

Methods Intended to Determine Traces of Albumin.

As there are many such methods, we will only describe those which seem to us to be of value.

This review may, at first sight, seem useless. but I believe that on reflection its value will be apparent.

Albumin, indeed, (a point which unfortunately is too little recognized, but which to me is incontestable) is not always precipitable, even in instances where the disease in its symptomatology is characteristic. There results this highly important fact: We should not rest content with a single test, but should always make control tests by other reagents; by following such a course we will often determine albumin where otherwise it would not have been noted.

This point established (and this is our justification for the details, possibly too technical, on which we enter) let us examine the various reagents recommended for the detection of small amounts of albumin.

Acetic Acid and Sulphate of Soda.—Into the albuminous urine dilute acetic acid is poured to a strong acid reaction, and then an equal portion of a saturated solution of sulphate of soda is added; the upper part of the mixture is next heated to boiling. The albumin is coagulated.

This method, which was recommended by Méhu,[14] gives excellent results; by means of it, indeed, an appreciable cloudiness of the urine is obtained when heat and nitric acid reveal nothing. In certain cases, however, as has been clearly proved by Bretet[15] of Vichy, this method does not reveal albumin which heat and nitric acid will. The method of Méhu, however, should not therefore be rejected, for it is certainly more sensitive than the usual reagents; the *modus faciendi* need only be modified. Méhu's method should first be used; if the result is negative add to the hot urine fifteen to twenty drops of nitric acid; a slight cloudiness will then appear, which will increase as the urine cools.

This double test is absolutely indispensable in order to affirm positively the absence of albumin. We will take occasion here to lay stress on the fact, again, that it is essential to test the urine in various ways.

Acetic Acid and Ferrocyanide of Potassium.—As in the former method acetic acid is poured to a strong acid reaction, and then a few drops of ferrocyanide of potass are added; in case albumin is present it is shortly precipitated in a flocculent form.

Acetic Acid and Phenic Acid.—To the urine is added three per cent. of nitric acid and ten per cent. of the following solution of phenic acid:[16]

Acid: Phenici	āā 1 part
Acid: Acetic (commercial) .	
Alcohol (90°)	2 parts

Shake and let settle.

Aceto-picric Acid.—The aceto-picric reagent is prepared by strongly acidifying with acetic acid a saturated solution of picric acid in distilled water. The urine is poured into a test tube and the acid is added drop by drop until no more precipitate forms; heat to boiling for some minutes.[17]

Picric Acid.—Into a test tube containing a concentrated solution of picric acid the filtered urine is poured drop by drop.[18]

If a cloudiness at once appears and this does not dissolve on shaking the tube, then we are dealing with albumin;

If the cloudiness is slight and not instantaneous it is usually due to the precipitation of uric acid (sometimes to the precipitation of certain medicinal alkaloids). This will be prevented if, before adding the urine, we heat the reagent slightly.

Chlorhydric Acid and Chloride of Sodium.—When the suspected urine is highly colored it is well to use the following reagent, which is highly spoken of by Roberts:[19]

Acid: Chlorhydric (D. 1.052). .	1 volume
Aquæ.	16 volumes

M.

With this mixture a saturated solution of chloride of sodium is saturated and filtered; the reagent is then ready.

Pour the saline solution into a test tube and add the urine; the presence of albumin is indicated by the formation of a cloudiness at the point of contact of the two fluids, and this cloudiness disappears on the addition of water; the albumin never coagulates.

Bichloride of Mercury.—A few drops of a 100 per cent. solution of bichloride of mercury is poured into the urine (an excess will redissolve the albumin) and heat; very rapidly a precipitate is formed which increases on boiling.

Bichloride of Mercury, Iodide of Potass and Acetic Acid.—Tauret[20] has advised the following reagent, which bears his name:

Iodide of potass	2.32 grammes
Bichloride of mercury . .	1.35 "
Acid: acetic	20 c. c.
Distilled water q. s. ad . .	64 c. c.

M.

This reagent is poured into the urine. A cloudiness forms. Heat and the precipitate remains, or else a granular precipitate forms which, after awhile, collects at the bottom of the tube.

This reagent, used to a great extent in France, has many disadvantages on which Professor Jaccoud has dwelt, with justice, at his clinic at Pitié:[21] "Many things are precipitated besides albumin." Indeed the reagent precipitates alkaloids and peptones; although these disappear on heating (a characteristic which differentiates from true albumin) it is better not to use this reagent; for, as Jaccoud says, "this reagent is dangerous, for the reason alone that it is essential to control it by other tests."

Such are the chief reagents recommended for the determination of albumin. What is their respective value?

G. Oliver[22] and Tyson, in a communication to the Philadelphia Medical Society, have tried to answer this question. The following, in a few words, are their results:

	Parts of liquid.
Sol. of picric acid revealed 1 part albumin in	20,000
Acidulated sol. of chloride of sodium " "	12,000
" " ferrocyanide of potass " "	10,000
Nitric acid " "	7,000
Heat " "	6,000

Let me add that, according to Tyson, heat combined with nitric acid reveals amounts of albumin inferior to those obtained by the use of one or another of the other agents alone.

Their sensitiveness is thus very great, especially that of the solution of picric acid; unfortunately it exposes to a number of errors which it is important to recognize.

The picric acid solution may redissolve the albumin when it is abundant; it yields a precipitate in the urine of patients taking quinine; it precipitates peptones.

Therefore it should not be used alone.

We see, then, finally, once again, that we ought always,

before affirming the presence of albumin, to make comparative tests with various reagents.

Determination of the Nature of Urinary Albumin.

It is not to-day sufficient to determine albumin, it is also essential to find out the variety of albumin which is present in the urine.

This question of the determination of urinary albumin is really of recent date, although it has been studied for fully thirty years.

Mialhe[23] was the first, as far as we know, to prove the existence of three kinds of albumin, each characterized by the nature of the albuminous matter in the urine.

We may find: *a.* Normal albumin, coagulating perfectly by heat and nitric acid, and not redissolving in an excess of acid; *b.* Modified albumin (amorphous or caseiform) imperfectly coagulated by heat, and forming with nitric acid a precipitate which is redissolved in an excess of acid. "This second variety," he adds, "is formed particularly in Bright's disease"; *c.* Albuminose, coagulated neither by heat nor by nitric acid, but by tannin, by creosote, by alcohol, by salts of silver and of mercury.

Mialhe's opinions are stated in two theses presented to the Faculté de Médécine, that of Dr. Mozian[24] and that of Dr. Ducom;[25] both insist on this little recognized clinical fact, and urge that the different kinds of albumin in the urine be sought for with greater care.

Nevertheless, the question was neglected in the years which followed, even by the writers who studied in particular albuminuria. In looking over the excellent article which Gubler wrote on this subject in the *Dictionnaire des Sciences Médicale,* 1865, we find that there does not exist a single kind of albuminuria. "This agent," says Gubler (page 516 and 517), "varies according to the case, and the urine in the same individual may contain various forms united. Some coagulating by heat and nitric acid, like egg and serum-albumin, others by heat and nitric acid together, and redissolving in an excess of acid, others finally not coagulable by heat but precipitable by nitric acid. Still," he adds, "the determination of these different kinds of albumin has as yet no clinical importance,

since not one is specially attached to Bright's rather than to other true albuminuria, and since albuminoid agents are readily transformed the one into the others under the influence of purely accidental causes."

Gerhardt[26] first proved that in urines not coagulable by heat and by nitric acid, a precipitate sometimes followed the addition of absolute alcohol due to the transformation of serum-albumin into peptone; he gave to this precipitate the name of latent albuminuria.

Next the chemist Lehmann[27] showed that by passing a current of carbonic acid gas through an albuminous urine a precipitate would form, having all the characteristics of globulin. These experiments were fully confirmed by Edfelsen[28] and by Senator,[29] who endeavored to find in albuminous urines all the albuminoid elements of the blood, as also Fuhry-Suethlage,[30] who with the same end in view performed a number of interesting experiments by dialysis.

It is only of late years that this question has been envisaged in our country.

Professor Lépine, of Lyons, was the first in France to study the albuminoid elements in urine; in 1880, by his assistance, appeared two theses containing the results of the experiments made at the Lyons school, the one written by Dr. Dauvé[31] and the other by Dr. Estelle,[32] and they proved that there were two varieties of urinary albumin: serin and globulin.

At the end of the year 1880 Charcot,[33] in his lectures on pathological anatomy at the Faculté de Médécine, said that in albuminous urine might be met, aside from serum, albumin or serin, globulin, and even peptones, as had long before been claimed by Gerhardt, and as had been confirmed by the experiments of Maixner[34] and by Hofmeister.[35]

Next, Flavard[36] and Faveret[37] finished the study of globulin; Nourry[38] and Wassermann[39] considered the question of peptones, which had been so thoroughly studied in Germany by von Jacksch.[40] Recently, Professor Jaccoud[41] has insisted particularly on the importance of searching for and of specifying the different kinds of albumin in the urine and has described at considerable length the different procedures to be followed in this direction.

Shortly afterwards Dr. Bengines-Corbeau[42] of St. Michel (*sur* Aisne) published a biological chemical study giving a complete *résumé* of the question.

Finally last year there appeared about the same time in France and in Italy a review of the clinical value of the different varieties of albumin in the urine.[43]

The fact then is perfectly established to-day that there does not exist a single kind of albuminuria; but that there are albuminurias.

The Different Kinds of Urinary Albumin.

"Making every allowance for further investigation (for the subject is not as yet completely settled) we may say to-day that there are three varieties of albumin to be considered in pathological urine."[44]

This expression of Jaccoud's in 1885, which was an echo of what Charcot said in 1880, remains perfectly true; there are three principal varieties of albumin in the urine: serin, globulin, and peptones; there are others but little known as yet and on which we will not spend time, except in regard to hemi-albuminose or propeptone, the history of which is about emerging from chaos.

Serin, identical with the serum of the blood, forms the most important of this group of substances. Albuminoid in nature, because it is constantly present in albuminous urines; in forty-one analyses of urine from renal affections made in 1876 by Petri[45] of the Berlin clinic, it was found forty-one times.

Serin, "the most pathological of the albumins, the Bright's albumin, *par excellence*" (Jaccoud), presents all the characteristics of albuminoid substances; it is analogous to egg-albumin; it differs, however, in the facts:

It gives no precipitate with ether; while egg-albumin precipitates in large lumps.

It redissolves completely in an excess of nitric acid; and this is not the case with egg-albumin. This latter fact it is important to remember, for it allows us, by the simple examination of the urine, to formulate the diet proper to the patient.

Globulin is far less frequently found than serin in albuminous urines (thirteen times out of forty-one; Clin., Berlin).

This albuminoid substance forms the most common type of the large family of globulins which to-day, according to chemistry, contains a large number of albumins formerly considered to be distinct species: dissolved fibrin, fibrino-plastic sub-

stance, casein of serum, hydropisin, crystalline, etc. Although its true name is paraglobulin (Kühne), it is commonly known as globulin (Weyl and Hoppe-Seyler); it has received this name because allied to hematin it constitutes principally the substance of the blood corpuscles.

Globulin presents the common characters of serin, coagulation by heat and by nitric acid; but it possesses also certain characters peculiar to itself which it is important to know:

It is more diffusible than serin; it passes through membranes with a facility equal to white of egg.

It is converted into concrete fibrin when, in contact with the air, it is mixed with a solution of fibrinogen.

At the end of three to four hours it becomes flocculent under the influence of a current of carbonic acid gas; but it redissolves when air or oxygen is introduced into the mass.

It is precipitated from cold urine when this is supersaturated with crystallized sulphate of magnesium.

The third variety of urinary albumin, described by Mialhe under the name of albuminose, of latent albuminuria by Gerhardt, and latterly by Lehmann under the name peptone, occupies by its frequency the second place in the urinary albumins, as witness the statistics cited twenty-eight times out of forty-one—that is, about two-thirds of the cases.

Of late years many monographs on peptones have appeared: in Germany, Maixner,[46] Hofmeister,[47] von Jacksch;[48] in Italy, Pietro Grocco,[49] Lussana,[50] Mya,[51] Primavera;[52] in France, Henninger,[53] Nourry,[54] Wassermann,[55] Georges,[56] Bouchard,[57] and they are differentiated from the other albuminoid substances in the urine by the following characteristics:

They do not coagulate by heat and acetic acid, nor by nitric acid, nor by the acetic solution of ferrocyanide of potassium, nor by any reagent ordinarily used in the tests for albumin.

They are coagulated by absolute alcohol as a flocculent precipitate which is redissolved by water, even though the masses have remained long in alcohol.

There remains a third urinary albumin; this is hemialbuminose or propeptone. Although far less known than the preceding, it forms a substance intermediate between ordinary albumin and peptones. As described by various writers, Salkowsky[58] and Lepine[59] for example, it has certain characteristics by which it may be recognized:

It does not coagulate by heat.

It coagulates when cold by nitric acid, by ferrocyanide of potassium and even by chloride of sodium in excess, in the presence of acetic acid.

Serin, globulin, peptones, propeptone, such are the principal varieties of albuminous substances which may be met with differently commingled in the urine of Bright's disease. Now that we have determined their distinguishing traits, it is time to expose the different ways of distinguishing and of differentiating them the one from the other, and we will be obliged to consider this at length, for their study is as yet hardly classical, and their determination often very delicate.

Means of Determining and of Differentiating the Various Kinds of Urinary Albumin.

Whenever we suspect the presence of albumin in the urine it is necessary (after having eliminated mucin, and after having verified its reaction) to test by heat and by nitric acid; under this test, according to the case:

There forms a precipitate. No precipitate forms.

In case of the first instance it is necessary to determine the nature of this precipitate, which may be due to serin, or to globulin, or to both simultaneously.

In the second instance it is necessary to prove by different tests that there is really no precipitate, that is, to search for peptones.

(Since hemialbuminose is rarely met with in the ordinary analysis, as a rule, we may dispense hunting for it.)

Precipitate under the Influence of Heat and of Nitric Acid.

What is the nature of this precipitate? Is it formed by globulin or by serin, or by both these albuminoid substances? Such are the questions to be resolved necessarily at the outset.

Globulin.—We should always begin by hunting for globulin. Its presence may be suspected when the urine is cloudy and does not clear on rest or on filtration, and especially when by the addition of water the cloudiness increases; but these

characteristics are far from sufficient to warrant us in affirming its presence.

To detect it three chemical procedures are at our disposal:

Pass a current of carbonic acid through urine containing globulin; at the expiration of three to four hours the urine becomes cloudy; at the end of one to two days (not always, however) a milk white precipitate forms which is redissolved on the addition of one per cent. chlorhydric acid and a few drops of marine salt.

This method is a very good one, but it is hardly adapted to clinical purposes.

Add a concentrated solution of chloride of sodium to the urine and a precipitate is formed if globulin is present.

This method has the advantage of being expeditious, but it has the disadvantage of giving no result when the amount of globulin is small.

The third method, that of Hammarsten,[60] is adopted uniformly, and in it the sulphate of magnesium is used.

We may proceed in two ways:

The urine is supersaturated with sulphate of magnesium, perfectly pure, and especially free from sulphate of iron; this salt being soluble in urine to an equal weight (80 per cent.), it should be dissolved by shaking until a final fragment remains undissolved. At the end of a few hours the globulin contracts into flocculent masses which need only to be filtered.

Add to a given quantity of urine (10 to 20 c.c.) an equal amount of a saturated solution of sulphate of magnesium (a few crystals should remain undissolved); then expose the mixture to cold for twenty-four hours. The globulin forms a coagulum which floats in the liquid or at the surface; rarely it precipitates to the bottom; the liquid may be decanted or filtered.

This method, which we habitually use, is very sensitive; it allows us to detect the slightest trace of globulin, even where the heat test reveals nothing in the urine.

Latterly Professor Ott,[61] of Prague, has said that when globulin is precipitated by the sulphate of magnesium, a portion of the serin is also thrown down if the urine contains a certain amount of acid phosphate (.5); therefore he recommends, with emphasis, that the urine be neutralized as much as is possible before the addition of the sulphate of magnesium.

To complete this question let us add that recently Pohl[62] recommends as a test for globulin the sulphate of ammonium, a test suggested some years before by Hofmeister; the following is the procedure:

Acid urines are neutralized with ammonia, and the deposited phosphates are filtered; we thus obtain what is essential —an alkaline urine. Add to this urine a saturated solution of sulphate of ammonia. In case globulin is present, at the end of an hour a precipitate is formed.

Although Pohl's method is much more rapid than Hammarsten's, we prefer the latter; it seems to us more reliable. In certain instances, indeed, we have thus detected globulin which the sulphate of ammonia had failed to reveal.

Serin.—Serin is sought for in the filtrate free from globulin; that this latter point may be assured special filter papers should be used (Berzéliu's filters); if this precaution be neglected globulin, which is a ready dialysate, will remain in the filtrate.

In order to detect serin in the filtrate, one of the methods already spoken of may be employed;[63] if a cloudiness or a precipitate forms it is due to serin; it may be isolated by filtration.

The urine, thus successively tested, should be absolutely clear of globulin and of serin—that is to say, of all the albuminoid substances which are precipitable by the agents ordinarily used in the search for albumin.

To be certain of this, and to avoid all chance of ulterior error, control tests should be made. We have a number at our disposal to which we will briefly refer:

Acetic Solution of Ferrocyanide of Potassium.—The urine acidulated by acetic acid is kept in a water-bath for several hours. The albuminous coagulum having been filtered, an acetic solution of ferrocyanide of potassium is added, and the mixture is allowed to rest for two to three hours. If at the end of this time the urine is perfectly clear all the albumin has been eliminated. If, on the contrary, there is the least cloudiness, albumin is present.

In the latter event, it is necessary to begin over again, to boil again the urine acidulated by acetic acid, and then to test, as above, the filtrate by the acetic solution of ferrocyanide of potassium.

This method is very tedious, for usually we are obliged to

repeat it; further, in certain cases, where the urine is too acid, or where it is primarily alkaline, no result is obtained. We therefore only mention this method in order to reject it absolutely.

Concentrated Solution of Acetate of Soda.—This method, advocated by Hofmeister, is far superior to the preceding in rapidity; Professor Jaccoud recommends it in his lectures.

To a half litre of urine 10 c.c. of a concentrated solution of acetate of soda is added, then perchloride of iron, drop by drop, until the liquid presents a permanent blood-red color. Next boil until the iron precipitates as basic acetate carrying with it the coagulated albumin. Then filter and in the filtrate not a trace of albumin should remain.

If we are to believe Wasserman [64] this method is not always precise; if it be repeated in order to separate traces of albumin, this may be transformed into hemialbuminose and even into peptones.

Chloride of Sodium.—Salkowsky [65] and latterly Dumonthiers [66] have lauded the following method:

Boil the urine previously acidified by acetic acid; add an excess of chloride of sodium; filter to clear the urine of the coagulated albumin.

This method, aside from its great simplicity, has the great merit of totally eliminating the albuminoids, as we have often pointed out; it is, therefore, the one which we have always used.

No Precipitate by Heat and Nitric Acid.—When there is no precipitate on heat or nitric acid, or rather when none forms, we should, after having resorted to one or another of the control tests enumerated, test the urine for peptones.

Many methods have been indicated with this end in view. Hofmeister ranks them under four; we divide them into two chief classes:

Methods for directly testing for peptones.

Methods for indirectly testing for peptones; that is to say, after precipitation.

Methods for Testing directly for Peptones.

These methods are simple enough, but unfortunately they are only precise when the peptones are present in definite amount, and when the urine is very slightly colored, or, better

still, colorless; although the latter condition is usually easy to meet, the same does not hold true of the former.

Whatever the case, we must proceed as follows:

Clear the urine of mucin and of albumin; then test the urine for peptones by Millon's reagent, or, preferably, by the reaction of bicyanate of ammonium.[67]

Millon's reagent, one of the most sensitive means for revealing the presence of all proteid bodies, whether in solution or not, is obtained as follows:

In concentrated azotic acid (density = 1.41, boiling point = 115 to 120°) dissolve mercury, at first by cold and then by gentle heat; at the end of an interval decant the clear liquid from the crystalline precipitate; the liquid thus obtained is Millon's reagent.

To use it add it to the suspected liquid and heat to 60° or 100°; if peptones be present we obtain a deep-red color which does not disappear on long contact with air or by protracted boiling; this color, much more accentuated than that produced by contact with other albuminoid substances, is absolutely essential for the affirmation of the presence of peptones (Maixner).

Far more preferable seems to us the *reaction du biuret,* bicyanate of ammonium test: add to the urine two drops of soda or of potass solution, and then a few drops of a two per cent. solution of sulphate of copper; if peptones are present, we obtain a coloration varying from violet-rose to purple-violet, when the proportion of peptone is considerable, although some writers claim that the shade of the color depends on the concentration of the solution and on the quantity of copper salt added to the liquid.[68]

This reaction, called *du biuret,* receives the name because the bicyanate of ammonium (*biuret*) acts in the same way in the presence of the same reagents; it occurs in the presence of other albuminoid matters, but the coloration is different; thus in a solution of serin a clear blue color is obtained.

Methods for indirectly Testing for Peptones.

In order to indirectly test for peptones—that is to say, after precipitation—alcohol, tannin and phosphotungstic acid may be used.

Aside from precipitation by alcohol, which is very defective, and precipitation by tannin, which requires too much time to be of value for current work, we will simply describe the method of precipitation by phosphotungstic acid:

Add to the urine $\frac{1}{10}$ of concentrated chlorhydric acid; next add a solution of chlorhydric of phosphotungstate of soda, the formula of which is:

Phosphotungstate of soda, ...	25 gr.
Chlorhydric acid, ...	5 "
Distilled water, ...	250 "

until there is no further precipitate. Filter and wash the filtrate with water acidulated by sulphuric acid three to five per cent.; heat for a short time. The peptone dissolves in the water, and filter to get rid of the insoluble barytes; there remains a colorless watery solution of peptone which is subjected either to Millon's, or, better still, to the *biuret* reaction.

This method, which according to Hofmeister reveals 25 gr. of peptone in a litre of urine, has been used by von Jacksch in all his researches on peptonuria.[69]

After his experiments, Hofmeister[70] proved that the solution of chlorhydric of phosphotungstate of soda precipitated creatinin. He, therefore, advised the substitution of the acetic solution of phosphotungstate of soda; if, following this advice, we employ the latter, the urine must first be acidified by acetic acid. In using this method we may consider as peptones any precipitate forming within five minutes; only the peptones precipitate thus quickly.

In addition to this method, which allows of the recognition of even traces of peptones, but which has the disadvantage of requiring much time, there are others less sensitive but more rapid; they must hence be mentioned.

Picric Acid.—This method has been highly lauded by Johnson,[71] professor of clinical medicine. A saturated solution of picric acid precipitates peptones as well as serin and globulin; it is, however, easy enough to differentiate the precipitates. Heat them and filter while hot; the peptones are dissolved and pass with the filtrate, while albumin (serin and globulin) remain on the filter-paper. The filtrate, subjected to the *biuret* test, should in case of peptonuria, give a color varying from rose to violet-purple.

Tauret's Reagent.—Even as picric acid so does Tauret's reagent precipitate the three principal albuminoids of the urine; serin, globulin, peptones; similarly, also, the precipitates act differently according to circumstances; the albuminous precipitate (serin and globulin) increases by heat, the peptonic precipitate disappears. We proceed as follows:

Into a test tube pour the urine entirely freed from albumin, and then add a few drops of Tauret's reagent; in case peptones are present, a cloudiness or a whitish precipitate forms at once. Heat the tube and the liquid clears. Plunge the lower part of the tube into cold water and the precipitate reappears, but only in the chilled part.

This property of disappearing in heat and of reappearing in cold is also common to the precipitate formed by alkaloids; therefore we must always, before affirming the presence of peptones, be assured that the patient has not ingested any substance of this nature during the days preceding the examination. Notwithstanding, it is well to use control tests, in order to differentiate between peptones and alkaloids; and alcohol readily discolors the latter and is without much effect on the former. Tauret's reagent would be then an excellent one for revealing peptones if it did not precipitate other substances; creatinin, guanin, xanthine, pus, semen, epithelial *débris*[72]; nevertheless, it may be used but only by resorting to control tests.

Before finishing this second part of our chemical study, let me say a few words only on the test for hemi-albuminose.

The urine having been freed from albumin add a neutral salt to saturation, and acidify with acetic acid; a precipitate forms which, subjected to the influence of heat, disappears completely, but reappears on cooling.[73]

The Dosage of Urinary Albumins.

Now that we are familiar with the methods of recognizing albumin in the urine and of testing for the different varieties, we must for completeness' sake consider a final question: the dosage of the diverse urinary albumins.

Dosage of Albumin.—By means of the precipitate yielded by heat or by nitric acid we may at once form an approximate idea of the amount of albumin contained in the urine,

and consequently of the absolute amount if we know the quantity of urine secreted in twenty-four hours. In the strongly albuminous urines the precipitate is flocculent, it is furfuraceous in urines moderately charged with albumin, and simply molecular in those which contain only a feeble quantity of the coagulable element.

The measure of the volume of the coagulum does not give much more exact indications than the simple inspection of the precipitates at the time of their formation. The particles resulting from the coagulation of a urine poor in albumin precipitate more slowly and do not tend to cohere to any great degree; they always occupy, therefore, a greater space than the larger and heavier flocculi which cling more forcibly together, so that the differences in height are far from expressing the differences in weight of the precipitates. Further, in case of the same urine, the deposit will vary according to two circumstances:

The duration of the boiling: the albuminous masses running together and enlarging according to the length of time during which the specimen is boiled.

The interval of rest: during which the liquid can separate and the precipitate can condense.[74]

For purely approximative determinations, for example, where the physician wishes to know if albumin is present in scanty or in large amount, or further if this quantity enlarges or diminishes, we may follow the annexed very simple procedure:

Precipitate the urine, by heat or by nitric acid, in a test tube of the same diameter throughout, and let it settle for from twelve to twenty-four hours; we may then readily compare the volume of the precipitate with the quantity of urine taken for test. By keeping the precipitates taken each day we may readily compare the varying quantity of albumin and thus see whether it increase or diminish. This method of testing is all the more exact if we take graduated tubes of exactly the same volume.

By this procedure we estimate only the relative amount of albumin contained in the urine.

Further this method is always a trifle inexact. According to the specific gravity of the urine, according as under the influence of heat the albumin has precipitated in small or in large

particles, the albuminous coagulum occupies now a larger and now a smaller volume. Vogel,[75] comparing this method with that by weight, has found errors of from thirty to fifty per cent.

This simple method is therefore inexact enough, even for judging the approximate quantity of albumin, and its results are quite incomplete. Therefore in order to obtain results perfect enough for clinical purposes we must resort to the method of dosage.

Many procedures have been devised for this purpose; we will only mention the principal, insisting on those which seem to us to be of value clinically. To pass them even rapidly in review, it is necessary to class them in groups, and we adopt the classification of Professor Lépine,[76] to be found in his notes to Bartel's treatise on Diseases of the Kidneys.

Densimetrical Methods.—The methods recommended by Lang,[77] Höebler[78] and Bernhardt[79] are at first sight seductive enough; they aim at obtaining the specific weight of the urine before and after the coagulation of the albumin; but, since it is very difficult to remove all the precipitate, and nothing but the precipitate, the results are very inexact.

Methods of Dosage by Opacity.—The aim is to determine the degree of cloudiness given by the coagulation of the albumin; in order to well appreciate this it is necessary, generally, to dilute the urine notably with a certain quantity of distilled water. The methods advocated by Vogel[80] and by Potain[81] are applicable especially to urines containing but little albumin, and they are exact enough if we are to accept the control results of Vogel and Masing.[82]

Polarimetrical Methods.—Biot[83] and Bouchardat[84] first determined that albumin deviates the needle to the left 50°; and Becquerel[85] having found that the degree of deviation was proportionate to the concentration of the solution, proposed this method for the dosage of albumin; according to him each degree will correspond to 18 centigrammes of albumin.

Although this method is excellent for determining the amount of albumin in water it is far from giving as good results in an albuminous urine; the coloration, the greater or less cloudiness interfere with the examination; the presence of salines renders equivocal the quantitative valuation of the

albumin; and therefore we often find quite great disagreement between the scales and the polarimeter.[86]

Methods of Dosage by Standard Solutions.—The best known is that of Bodeker,[87] which gives very good approximate results; and the same holds true of Tauret's[88] method, which is more rapid in its results.

Methods of Dilution.—These methods are based on the fact that if the quantity of albumin is very slight, and nitric acid be added with care, the albumin ring only appears after the lapse of some minutes.

Roberts[89] and Stolinkoff[90] dilute the urine by degrees until the albumin ring only appears at the end of about forty seconds. Brandberg[91] follows the same course, only he dilutes more, so that the ring only appears at the end of three to four minutes.

The method of Musculus[92] is somewhat different; instead of varying the dilution of the urine he takes account of the variable time required by the albumin ring in forming in a urine diluted enough to require a certain time before this ring should form. This writer has prepared a table which may be consulted to determine at once the richness in albumin of the urine examined. Here are a few of the figures:

Quantity of Albumin to the Litre.	Beginning of the ring.	Ring well formed.
0 gr. 20	at once	½ minute
0 gr. 10	½ minute	1 "
0 gr. 05	1 minute	2½ minutes
0 gr. 01	7 minutes	15 "

This method, which is attractive enough, has been used several times by Professor Lépine who was not specially pleased with it; it is hardly likely that it can be used clinically.

Method of Redissolution.—This method has very lately been advocated by Dr. Luton[93] of Reims.

By means of heat the albumin in a known quantity of urine is coagulated, and it is then redissolved by a standard solution of tartaric acid.

Up to now Dr. Luton has only been able to obtain relative results; he hopes, however, in the future, to obtain absolute results—that is to say, to determine the exact weight of albumin contained in each litre of urine.

Method by Weight.—Weight in the dry state of the coagulated albumin is without question the most exact method of dosage; on this point all chemists are in agreement, only differing in regard to the substance to be used for coagulating the albumin.

Some prefer heat, the urine having been first acidified by acetic acid; others alcohol; in the first instance precipitation is incomplete; in the second other substances are precipitated at the same time as the albumin. This is the reason why Méhu[94] recommended phenic acid; this acid combining with albumin, we are exposed to an error which may reach sixteen per cent. Esbach,[95] who determined this point, rejects this acid absolutely, and recommends precipitation of the albumin by picric acid; the picrate of albumin thus obtained is weighed and the weight is multiplied by .8. In order to place this method above reproach it is necessary (and this is by no means proved) that the albumin and the picric acid should always combine in the same proportion.

An excellent and rapid method is for certain chemists the bi-chloride of mercury, but this salt unfortunately precipitates in part the peptones.

Whatever the coagulating substance used this method is liable to errors which we must remember if we desire to obtain exact dosage; further it takes time and great care. It seems to us, therefore, scarcely adapted to clinical purposes, and this is the reason why we do not describe it at length.

Methods by Deposit.—There remain to be considered the methods which consist in measuring the weight of the albuminous deposit in a tube where the albumin is precipitated always from the same quantity of urine.

Although the method is about identical in all the instances, it is not in regard to the coagulating agent used; nitric acid,[96] phenic acid,[97] picric acid,[98] these agents have been recommended; the latter, in particular, especially as recently modified by Esbach, seems to give excellent results. We will, therefore, describe it somewhat at length; and it is the method ordinarily resorted to.

The following is the principle on which it is based:

In a graduated tube the albuminous urine is treated in a cold solution of citro-picric acid; after an interval of rest read the height which the coagulum attains. Since the graduation

of the tube represents in grammes the amount of albumin contained in a litre of urine, we know at once the amount of albumin in the specimen examined.

Special reagents and graduated glass tubes are two elements necessary. We will say a few words of these:

To obtain the reagent, proceed as follows:

In 600 to 800 gr. of water, dissolve by heat 10 gr. picric acid and 20 gr. citric acid, pure and dried in the air; on solution add a quantity of water sufficient to make a litre, such is the citro-picric reagent (modified) of Esbach. (Picric acid precipitates albumin, while citric acid keeps the phosphates and the urates in solution.)

The graduated tubes are .15 cent. in height and .015 millim. and ½ interior diameter. Above the line of grammes is marked a U, and higher up an R.

Proceed as follows:

Add the urine up to the U, and the reagent up to R, stop the tube with the thumb and turn the tube twelve times without shaking (each turn including the inversion and re-inversion of the tube). Close with a rubber cork and allow to rest for twenty-four hours. At the end of this interval, read the height of the coagulum, at the centre of the albuminous surface, which is ordinarily more depressed than the borders. In order to obtain satisfactory results by this method it is essential:

The Urine must be Acid.—Since albuminous urines undergo more rapidly than others ammoniacal fermentation, we must always assure ourselves by test paper of the acidity of the specimen. It must give a strong acid reaction.

The urine must not be too rich in albumin. If the urine appear too rich add one to two volumes of water, not exceeding four grammes total amount; take this dilution later into account by doubling or triplicating the obtained reading.

This method, say Neubauer and Vogel, is very simple, does not require much time, and gives exact enough clinical results.[99]

Guttmann[100] in a recent article reaches the same conclusion. He says: "Owing to the results obtained from Esbach's method, which are nearly as exact as those yielded by weighing the dried coagulated albumin, and owing to its ease of application, I cannot too highly endorse it."

The method of Esbach is, however, liable to error; it may yield a precipitate when there is not a trace of albumin in the

urine; this fact has recently been noted by Dr. Kuthe [101] and by Teil (of Holland) in a letter addressed to our teacher, Dr. Dujardin-Beaumetz, and we reproduce the criticism here:

In a patient taking the sulphate of quinine in quite large dose, Dr. Kuthe found by Esbach's tube a manifest precipitate, while he obtained none by the ordinary reagents. Supposing this precipitate to be due to the sulphate of quinine, he suppressed it and the precipitate could no longer be obtained; it was due then to the formation of the picrate of quinine.

This fact it is important to note; we must bear it in mind when examining the urine of patients taking quinine. In doubtful cases, we may readily enough differentiate the quinine precipitate from an albumin coagulum; the first disappears by heat, while the second persists.

We may conclude, then, that although Esbach's method does not suffice to determine traces of albumin, it allows us to follow efficiently the daily variations in amount; and it is hence of signal value in the routine examination of the urine of Bright's disease.

Dosage of the different Kinds of Urinary Albumin.

The dosage of albumin as an entity being such a relatively complex affair, when we desire perfectly exact results, it is readily apparent how much more difficult is the separate dosage of the different kinds of albumin; we will, therefore, deal briefly with this part of the subject, seeing, also, that it is as yet hardly fit for clinical purposes.

Globulin and Serin.—We will describe in his own words the method devised by Professor Lépine,[102] who has specially studied these substances:

"50 to 100 c.c. of filtered urine are mixed with an excess of sulphate of magnesium and allowed to rest; at the end of an interval small flocculi appear in the urine. Pour through a filter paper, the weight of which is known, and wash the precipitate with a cold saturated solution of sulphate of magnesium. The filter paper and the precipitate are inserted into a vessel with a small quantity of water and shaken during a period sufficient to reduce the paper to a pulp. The contents are next poured into a porcelain capsule, in order to render insoluble the globulin, which has been partially, perhaps entirely,

redissolved in the water; boil and pour on a filter paper, wash with boiling water, dry at 100°, and weigh with the usual precautions; deducting the total weight from that of the first filter paper we obtain the weight of the globulin. To obtain that of the serin, we use the first filtrate which has been freed from globulin; acidify it slightly, boil, recover the precipitate on a filter, wash with boiling water, dry and weigh."

Our object in entering into the details of this method of Lépine has been to prove its difficulties. Such a method is not at all applicable clinically; too much time is required, the manipulations are of too delicate a nature for routine purposes.

For this reason we prefer a method less complex, which allows us to differentiate the weight of the globulin. We proceed as follows:

Divide the filtered and acidified urine, which has been freed from mucin, into two parts.

Treat the first part by the citro-picric reagent in an Esbach tube; on the following day we know the total quantity of albumin, that is to say, the amount of serin and of globulin combined.

To the second part is added a saturated solution of sulphate of magnesium; on the following day globulin, if present, is precipitated; filter. The filtrate, containing only serin, is poured into an Esbach tube and the citro-picric reagent is added. In twenty-four hours a coagulum is obtained which represents the amount of serin by multiplying by two the figure obtained, since the liquid acted on the second time is a first dilution of the urine; it is, in fact, composed of half urine and half saturated solution of sulphate of magnesium.

Deducting the weight of serin from the total weight of the serin and the globulin we obtain the amount of globulin.

Such is our method; although the results are not pefectly exact, and this we readily grant, they are at least enough so to allow of comparison from day to day; the method is simple and clinically applicable to routine purposes.

Peptones.—Up to now we are satisfied with determining the presence of peptones; no one has as yet proposed a method of dosage. We have had no personal experience and therefore will not dwell upon the subject.

Method of Examining an Albuminous Urine.

Before ending these chemical considerations, we must state the steps we have followed in our various researches.

1. We first filter the suspected urine; if, after filtration, it is cloudy, we add a little acetic acid to precipitate the mucin; at the end of a few minutes this is filtered off.

2. We next verify the reaction of the urine; if it is not sufficiently acid—that is, if it does not redden litmus paper markedly—we add a few drops of acetic acid.

3. A part of the acidified urine is then subjected to heat or to nitric acid, to find out whether it precipitates or not:

In the first instance we seek the nature of the precipitate: To 10 or 20 c.c. of urine, rendered neutral, we add a saturated solution of sulphate of magnesium; at the end of twenty-four hours we obtain or not a precipitate or cloudiness which we filter. In the filtrate, which is deprived of globulin, we seek for serin by one of the methods we have mentioned.

In the second case, that is to say when no precipitate forms, we resort to a control test to eliminate the albumin (serin and globulin); these are completely separated by the chloride of sodium. We then seek for peptones by the biuret reaction, after having first precipitated them by the acetic solution of phosphotungstate of soda.

4. The remainder of the urine is subjected to Esbach's method; the first test gives us the total amount of albumin, that is, of globulin and serin. The globulin having been precipitated by the sulphate of magnesium and filtered, we test the filtrate again by Esbach's method; the obtained figure, multiplied by two (since the urine has been diluted once), represents the quantity of serin.

The difference between the figures obtained by these two procedures represent the amount of globulin.

We believe that these methods of determination of the albuminoid substances, although only approximate, are exact enough for clinical purposes; they allow us to take account of the daily variations of the globulin and the serin in the same patient.

5. We complete our analysis by noting:

a. The quantity of urine; *b.* The density of the urine; *c.* The amount of urea, calculating this only after the elimination of the albuminoids, which otherwise might lead to error.

Such are the methods, unfortunately rather tedious, to which we must resort in order to make a complete examination of an albuminous urine.

We thus only obtain approximate results, however, but sufficient clinically, for, as M. Gubler says:

"Clinically, absolute exactness is not indispensable."

BIBLIOGRAPHY.

1, Chémie medicale.—2, De l'urine et les sediments urinaires, p. 161, 1887.—3, Anl. zur. Harn., Wien u. Leipzig, 1881.—4, Clin. Méd. Charité, 1867, pp. 853, 854.—5, Bull. Thérap., p. 17, 1883.—6, Comptes rendus, Societé Biologie, 1884.—7, Dict. Dechambre, Art. Albuminuria. —8, Arch. f. Chem. u. Micros., 1852.—9, Trans. of Soc. for Improvement of Med. and Surg., vol. iii., 1812.—10, In Bengines-Corbeau, 1885.—11, Berlin. Klin. Woch., 1881.—12, Soc. Méd. Berl., 1883.—13, Brit. Med. Journ., 1883.—14, Chim. Med., 1878.—15, J. Pharm., et Chim., 1886.—16, Méhu, loc cit.—17, Capitan, Th., Paris, 1883.—18, Esbach, Bull. Thérap., 1880.—19, Lancet, 1882.—20, Bull. Thérap., 1877.—21, Clin. Med. Charité, 1885.—22, Lancet, 1883.—23, Mem. sur la Digest. et l'Assimil. des matiéres albuminoides, Acad. Sc., 1846.—24, De la Maladie de Bright, 1854.—25, Recherches sur les matières albuminoides, 1855.—26, Deutsch. Arch. f. Klin. Med., 1868.—27, Virchow's Archiv., vol. lvi.—28, Deutsch. Arch. f. Klin. Med., 1876.—29, Ibid., 1878.—30, Ibid., 1876.—31, Pathogenie de l'albuminurie.—32, Des matières albuminoides dans les urines albumineuses.—33, Progrés Med., 1880.—34, Prag. viert. f. pract. heilk., 1879. —35, Prag. med. Woch., 1880.—36, Dosage de l'albumine, Lyon Med., 1881.—37, De l'albuminurie, Th. Lyon, 1882.—38, De la peptonurie, Th. Paris, 1884.—39, Ibid., 1885.—40, Zeits. f. Klin. Med., 1883.—41, Clin. Méd. Charité, 1885.—42, Revue Ther., 1884-1885.—43, Chérm, Un. Med., 1886 and Rif. Med., 1886.—44, Jaccoud, loc. cit., p. 564.—45, Diss. inaug. Berlin, 1876.—46, Loc. cit.—47, Loc. cit.—48, Zeitsch. f. Klin. Med., 1883. —49, Della peptonuria, An-Univ. di Med. e di Chir., Napoli, 1883.—50, Della peptonuria, Riv. Veneta di Sc. Med., 1886.—51, Sur le cause della peptonuria, Gaz. Clin., Torino, 1886.—52, Peptonuria falsa e vera, etc., Groen. int. di Sc. Med. 1886.—53, De la Nature et du rôle physiologique des peptones, 1878.—54, Loc cit.—55, Loc. cit.—56, Etudes sur la peptourie, sa pathogenie, Th. Paris, 1886.—57, Des peptonuries, Un. Med., 1886.—58, Virchow's Archiv., 1873.—59, Notes additionelles au traité de Bartels, p. 550.—60, Jaccoud, loc. cit., p. 573.—61, Prag. Med. Woch., 1884.—62, Wien. Med. Woch., 1886.—63, Heat, nitric acid, acetic acid and ferrocyanide of potassium, etc.—64, Loc. cit.—65, Loc. cit. 1876.— 66, Th. Pharmacie, Paris, 1886.—67, Reaction de biuret.—68, Huppert, Prag. Med. Woch., 1880.—69, Hoffmeister: Zeitsch. f. phys. chem., 1880,

vol. iv. Von Jacksch; loc. cit.—70, Loc. cit. vol. v.—71, Brit. Med. J., 1883.—72, Méhu: Ann, Mal. Org. Gen. Ur., 1884.—73, Lepine: Notes add. au traité de Bartels.—74, Gubler, loc. cit., p. 17.—75, Neubauer and Vogel, loc. cit. p. 369.—76, Loc. cit., pp. 541-546.—77, Arch. f. Anat., 1868. —78, Arch. f. Anat., 1868.—79, Deutsch. Arch. vol. xvi. p. 222.—80, Zeitsch. f. Anal. chem., vol. vii., p. 152.—81, Bouchardat: Glycosurie. 1875.—82, Deutsch. Arch., vols. iii. & iv.—83, Bartels, p. 545.—84, Ibid.—85, Séméotiques des urines, etc., 1841.—86, Haas: Pfuger's Arch., vol. xii.—87, Ann. Chem. u. Pharm., vol. lxi.—88, Bull. Gen. Thér., 1877.—89, Lancet, 1876.—90, St. Pet. Med. Woch., 1876 —91, Analyse in Maly's Jahrerbericht, 1870.—92, Gaz. Med. de Strasbourg, 1870.—93, An. Med., Nord-Est, 1879.—94, Loc. cit., p. 63.—95, Bull. Therap., vol. i., 1880.—96, Hamm: Gaz. Hop., 1858, and Gaz. Med., Paris, 1861 and 1862.—97, Heminow: St. Pet. Med. Woch., 1879.—98, Esbach: Gaz. Med. de Paris, 1874, and Bull. Therap. vol. i., 1880,—99, Loc. cit., p. 286.—100, Berl. Klin. Woch., 1880. —101, Bull. Thérap., 1883,—102, Loc. cit., p. 548.

CHAPTER II.

THE DIAGNOSTIC VALUE OF THE SYMPTOM ALBUMINURIA, IN BRIGHT'S DISEASE.

BEFORE passing to the clinical side of this question we believe it useful to recall in a historical way the various phases through which this interesting question of albuminuria has passed. This digression appears to us a proper introduction to this chapter; it shows us, in fact, what was formerly considered the value of albuminuria, and it impresses on us what ought to-day to be its value.

HISTORICAL CONSIDERATIONS.

Discovery of Albuminuria.—Cotugno[1] first in 1770 determined that certain dropsical patients eliminated non-bloody urine which, when subjected to heat, gave rise to a coagulum similar to coagulated white of egg.

This discovery of Cotugno's was thirty years afterwards completely confirmed by the researches of certain English physicians: Cruiksank, Wells and Blackhall. Cruiksank[2] in 1798 based on the presence of albumin in the urine the principle of his classification of dropsies, dividing them into two chief classes: Albuminous dropsies, of general origin, and non-albuminous dropsies of visceral origin. This division was soon approved by his two compatriots. Wells[3] sometimes found at autopsy on individuals, who during life had had albumin in the urine, special alterations of the kidneys: "kidneys harder, with thickening of the cortex, alteration of the structure from the deposit of coagulable lymph, etc." He determined only the fact without drawing any deduction: "I do not thence conclude," he says, "that the kidneys are always diseased, when the urine contains much serum."

Blackhall[4] observed in an unusual proportion of cases renal lesions in dropsical and albuminuric individuals, but, doubtless

influenced by the alterations which he found both in the serous membranes and in other parts of the body, he saw in these diverse lesions the result of a general affection of an inflammatory nature. Indeed, while recognizing the validity of the observation of his predecessors, he was unable to determine the origin which remained unknown up to the well-known researches of Richard Bright.

Albuminuria and Bright's Disease.—The origin of albuminuria, Bright,[5] with reason, traced to affections of the kidneys, and he described very thoroughly the disease which bears his name. Before his day, as we have seen, it was known that dropsy was often associated with diseases of the kidney, and it was also known that certain dropsical patients had albuminous urine, but the frequent coexistence of dropsy and albuminuria with renal disease had never been insisted upon; this fact Bright established in the clearest possible manner in his first volume,[6] published in 1827. After having described the different causes of dropsy[7] he announces that dropsy may be due to a further cause, a special alteration of the kidneys, on which he dwells somewhat at length, and he adds that whenever dropsy depends on these alterations, the urine is more or less albuminous, while he has never found it coagulable in a large number of other cases of dropsy which he has examined. Convinced of the great importance of the examination of the urine in this new class of dropsies, he had the happy idea of invoking the collaboration of Dr. Bostock,[8] well known from his important physiological and chemico-pathological works. The latter (his experiments are related in Bright's work) determined a fact fully verified at least in great part, that in this new form of dropsy—

The urine always contains albumin, generally in considerable proportion, varying, however, according to the case.

The specific gravity is notably diminished. The proportion of urea and of salts sustains equally in all the cases a more or less pronounced diminution.

Bright's views were soon sustained in England by Christison[9] and Gregory.[10] Basing their views on numerous experiments these two writers concluded that we always find post-mortem renal affections in patients who during life presented albuminous urines.

A few years later, in France, Martin-Solon,[11] notwithstand-

ing the numerous facts noted by his contemporaries showing the diverse origin of albuminuria, defended the ideas of Bright, of Christison and of Gregory. In his book (1838) on albuminuria, he described various affections, independent of Bright's disease, but having the common characteristic of albumin in the urine.

Few authorities, it is true, accepted these ideas; they adopted, the majority at least, those which two years later Rayer promulgated in his excellent treatise on diseases of the kidneys.

Albuminuria as a Symptom.—Already, before the appearance of this French work, opposition had appeared to Bright's views.

In 1832, Spittal,[12] in his inaugural thesis at Edinburgh, while granting the views of the distinguished physician to Guy's Hospital and his successors, stated that they were a trifle too absolute, and that the presence of albumin was not always a certain sign of the renal changes described by Bright.

A year later there appeared in France by direction of Rayer, the first work on this question, the thesis of Dr. Tissot,[13] "On Dropsy caused by Granular Affection of the Kidneys;" he determined that the presence of albumin in the urine is a sign of great value in Bright's disease, but he does not grant it absolute value, for he had found albumin in many affections to which he briefly refers. This thesis was the starting-point of many writings, instigated largely by Rayer. The determination of albumin became a matter of routine in the hospitals of Paris, and soon there arose striking differences in the observations.

Some writers accepted as proved in their entirety the ideas of Bright; for them Bright's disease and albuminuria were synonyms; Martin-Solon (to whom we have referred) personifies this school, which, notwithstanding the facts, for many years had but few adherents.

Others, having found albumin in cases where post-mortem the lesions described by Bright were absent, and also in patients not at all dropsical, concluded that the presence of albumin was absolutely of no value, or at least of very little value, since albuminous urines were met with in many diseases different both in seat and in nature.

Others, finally, and the great majority it must be confessed,

while admitting that albumin was found in other affections, considered albuminuria as a good sign of Bright's disease, but claimed that the symptom did not alone carry weight, but must be associated with other precedent or accompanying symptoms. This was the keynote of Desir's treatise (1835): "On the Presence of Albumin in the Urine as a Phenomenon and a Sign of Disease." This was followed two years afterwards by the letters of Forget, Professor of Clinical Medicine at the Faculty of Strassburg, letters addressed to Rayer and published in the *Gazette Médicale de Paris.* We reproduce the following extract, which seems to us to give an exact enough idea of the state of the question at this period:

"In how far does the art of diagnosis possess unequivocal, infallible, pathognomonic signs, in a word? Now what holds true for other diseases, does for albuminuria; after having pretended that the symptom uniformly meant an affection of the kidneys, the cases will be specified and emphasized which are exceptions, as in case of every other symptom, but the fundamental fact will remain established even though it be not infallible, and science will still be able to record a precious victory."[14]

The same opinion was expressed by Bouillaud[15] and by Rayer, in his superb treatise on diseases of the kidneys;[16] in England by Ellioston,[17] by Graves,[18] by Copland.[19] Bright himself[20] recognized this fact; he rejected the exaggerations of his successors, and claimed that he had never completely identified albuminuria with renal lesion.

Although Rayer's views were not at once accepted by all, they were rapidly enough in the years following the appearance of his treatise, years during which numerous theses and important works endeavored to determine the different cases in which albumin might be found in the urine, and to specify the value of the symptom albuminuria.

Albuminuria as a symptom was then an established fact, and it is no wonder that the Faculté required as a thesis for the degree of doctor in medicine: "The symptomatic value of albuminuria." Montanier,[21] chosen to sustain this thesis, begins in the following striking words:

"Some writers have made the word albuminuria a synonym for Bright's disease, for albuminous nephritis; this error has not gained ground, and to-day for everybody the word albu-

minuria represents a symptom common to a variety of affections; this symptom is the passage of albumin, or, better still, of the serum of the blood into the urine."

Since that time albuminuria has ever been regarded as a symptom, but the number of affections in which albuminous urine has been found has greatly increased.

Albuminuria in Health.—The presence of albumin in the urine of perfectly healthy individuals was discovered by Bostock (Bright's work) in 1827, and was confirmed by Spittal (Th. Edinb.) in 1832; in his inaugural thesis, previously referred to, he affirms on the authority of Turner and de Gmelin, that albumin exists in healthy urine, since it may be precipitated by tannin and the bi-chloride of mercury.

Notwithstanding, this question was neglected for many years; only lately was it reconsidered, first in England and in Germany, and next in France.

Vogel [22] and Ultzmann [23] reported cases of albuminuria in perfectly healthy subjects, but it is Leube [24] who first studied physiological albuminuria; he examined the urine of 119 soldiers in active service and in good health, at different times of the day, in the morning, in the afternoon, after a long march, and at night, and he found:

5 times albumin in the morning (4 per cent.)
19 " " " afternoon (19 ")
Never albumin at night.

The quantity of albumin found was further never considerable, never exceeding one per cent.

Gull [25] and Dukes [26] made about the same time analogous experiments on infants in England; they obtained the same results as the German writers. The same holds true of Moxon [27] who found the facts holding in particular for anemic and debilitated infants.

In France, Lépine [28] who first turned his attention to this question, proved that in a large number of patients albumin was found, at certain hours of the day, even in rather large proportions; and he thence concluded that albuminuria should only be considered pathological when present in large amount and associated with deterioration of the general health. Soon after Capitan [29] and de La Celle de Chateaubourg [30] confirmed the frequency of this physiological albumi-

nuria (in 700 samples of urine the latter found albumin in notable amount in 592 cases), and proved the variations from day to day.

Recently, Senator,[31] in his excellent study of albuminuria, has admitted—a fact, however, also proved by Possner[32] and Noorden[33]—that all normal urine contains serum albumin, but in rather small amount (½ milligramme to the litre, according to Possner).

Although this fact, determined by Senator, is of slight importance, since such a small quantity is not determinable by the ordinary reagents, the same does not hold true of the facts determined by Lépine, Capitan, and de La Celle de Chateaubourg. In all the observations of these writers we find appreciable amounts of albumin in patients presenting all the characteristics of perfect health.

From what has gone before, then, there results the following positive fact: side by side with pathological albuminuria there exists a physiological albuminuria which deserves serious consideration.

Bright's Disease without Albuminuria.—The presence of albuminous urine in many and in diverse affections had considerably diminished the value of this symptom in Bright's disease; the frequency of albuminuria in patients in perfect health had contributed still further to diminish this value; nevertheless, albuminuria remained one of the diagnostic signs of Bright's disease. If albuminuria could exist without Bright's disease it seemed impossible that Bright's disease should exist without albuminuria.

This fact is to day, however, perfectly established, although by rather recent research. It was, if we are not in error, first called attention to in the thesis of Dr. Mozian:[34]

"Bright has the merit of having established the narrow corelation existing between the special lesion of the kidneys with albuminuria and the development of dropsies. Does it follow that these three phenomena form a species of trinity, one and indivisible? No, assuredly not. Ulterior observations have modified our ideas; dropsy may be lacking, and albuminuria, although constant, so far at least, in the renal lesion of Bright's, may be found under quite different circumstances."

A few years later Dr. Hue,[35] in his inaugural thesis is more explicit; he thus expresses himself:

"Whenever at autopsy we meet these anatomical lesions of the kidneys, we are sure that, during life, the urine has contained, for a greater or less time, albumin in greater or in less amount.

"Monneret and Graves have each reported a case in which the kidneys presented all the lesions of Bright's disease, and yet the urine had never offered the slightest trace of albumin. Since then, Mazomer, a Russian physician, has reported three additional observations of Bright's disease without albuminuria. We must admit that if many similar cases are recorded, albuminuria as a symptom will lose much of its importance. But such cases are exceptional.

"Are we not justified in asking if there be not erroneous interpretation, or if these cases may not enter into the following category: When we carefully follow the progress of Bright's disease, and we examine the urine each day, it frequently enough happens that, during a longer or a shorter time, and even several times during the course of the disease, no precipitate is obtained either by heat or by nitric acid. We may thus daily follow the diminution of the albumin, up to its complete disappearance, and then, after a variable time, it reappears gradually or very quickly.

"We see then that Bright's disease may exist without albumin being present in the urine; but this is dependent on the fact that the disease was not watched long enough. Whenever albuminuria is present and persists there is, without question, Bright's disease, but this may persist without albuminuria; it seems to us that the cases, of Monneret, Graves and Mazomer may readily be classed in this category."

We have made this long extract from Hue's work because it shows us well the state of the question at this period; it gives us also a just enough explanation of the fact. Still the possibility of Bright's disease pursuing its course without albumin in the urine was for long considered an anomaly. Of late years we find the question again studied. In 1874, Huchard,[36] in an article entitled "Quelques considerations sur les nèphrites latentes," insists particularly on this point—that nephritis, especially the interstitial form, may run a latent course; edema and albuminuria may be lacking, and the chronic inflammation of the kidneys may be accompanied only by persistent anemia, loss of flesh, digestive disorders, and special troubles of the intellectual faculties.

Lecorché[37] holds the same opinion; he recognizes the fact that in interstitial nephritis the urine does not necessarily contain albumin, and that this form of disease may pursue its course without our ever finding a trace.

The English and American writers are still more explicit:

Johnson[38] first studied this question, and he was followed by Mohamed; in a paper read before the London Congress (1881) and published *in extenso* in Guy's hospital reports,[39] the latter reports the cases of nephritis treated at Guy's hospital during 1879-1880; of 61 cases in 41 albumin was never found, and in 11 only at infrequent intervals. 21 of these cases died, and in all, post-mortem, renal lesions were found.

Therefore, according to him, neither albuminuria nor dropsy exist habitually, in chronic Bright's disease; when these symptoms appear they are the indices of acute or epithelial alterations.

The following year (1882) the same ideas were sustained by Millard in the New York Medical Journal: "On the Exclusion of Albumin in the Diagnosis of Interstitial Nephritis."

Soon in France the question was specially studied, first by Dr. Delespierre and then by our accomplished teacher, Professor Dieulafoy. In 1884 Delespierre[40] wrote in his inaugural thesis the following words:

"Albuminuria cannot be considered as a constant symptom in nephritis; often enough it may be wanting, and yet the clinician may find in the symptoms of the disease the elements for certain diagnosis."

More recently Professor Dieulafoy, who has for long studied specially the diseases of the kidneys, read before the *Société médicales des hôpitaux* (June 11, 1886) the report of four convincing cases in which he was able for a greater or less time to determine the complete absence of albumin, although many symptoms of Bright's were present, and ultimately autopsy proved disease of the kidneys. A few months later (October 22, 1880), in a second paper, he presented to the academy three further cases of Bright's without albuminuria. Finally, very recently, in his lectures at the School of Medicine, he has laid special stress on these cases of Bright's disease without albuminuria which it is so important to recognize. We have often personally during our term of service had the opportunity to testify to the exact researches of our teacher.

The fact, then, is perfectly established to-day that Bright's disease may exist without, during life, albumin being detected in the urine.

To resume, then, the history of this affection in a few words:

Albuminuria, first considered a symptom of but little importance, and as an anomaly, gradually became the pathognomonic sign of Bright's disease to such a degree that certain clinicians considered albuminuria synonymous with Bright's disease.

Next, numerous observers, having found albuminous urine in many and in diverse affections independently of all renal lesion, and again having found albumin in the urine of subjects enjoying the most perfect health, albuminuria became a symptom common to many diseases. This symptom, the absolute value of which has certainly diminished, has still, however, great weight in the diagnosis of Bright's disease. When a number of symptoms suggest the possibility of a chronic renal affection, albuminuria remains the great diagnostic criterium; the affection remains doubtful if this symptom be lacking, and it becomes certain if this symptom appears.

Finally, it became a certain fact that Bright's disease may exist for a greater or less time, perhaps for a life-time, without albuminuria; the symptom thus loses much of its diagnostic weight. If its presence (under certain conditions) still indicates truly a renal affection, its absence does not entitle us to reject the affection, and the diagnosis of Bright's disease may be made even when the most sensitive reactions do not allow us to discover the slightest trace of albumin in the urine.

Diagnostic Value.

Albuminuria, an unquestioned fact to-day, has lost to a great degree its old-time diagnostic value.

Does it still possess value? If so, to what degree? These are the questions we aim at answering.

We will begin by establishing the indications we are justified in drawing from the presence or the absence of this symptom.

We will next see if the physical and chemical nature of albuminuria can give us information in regard to its origin and its cause.

We will then examine if it is possible to draw the relations which exist between the presence of albumin in the urine and the urinary secretion,—considerations of interest in regard to the lesion.

We will conclude by seeking if there exist between albuminuria and the various symptoms presented by the patient, relations which will help us in affirming the presence of Bright's disease.

Having passed all these questions in review, we trust we will be in a position to answer the first question of our research:

What is the diagnostic value of albuminuria in Bright's disease?

The Existence of Albuminuria.—We cannot examine this question of the presence of albuminuria in its entirety; for the sake of ease of description certain divisions are essential; we must take clinical types; those with which we meet most frequently may be readily classed under the following three heads:

Parenchymatous nephritis.
Interstitial nephritis.
Mixed—or, better still, diffuse nephritis.

Although, as has been with justice emphasized by Dieulafoy, it is this latter form which is met with usually, we will dwell at greater length on parenchymatous and on interstitial nephritis, because they present more marked characteristics.

Parenchymatous Nephritis. — Albuminous nephritis (Rayer); epithelial nephritis (Lancereaux); tubular nephritis (Dickinson); non-desquamative nephritis (Johnson); large white kidney (Bright's kidney).

Presence of Albumin.—The capital alteration of the urine in this variety is the presence of albumin. This is of such importance that since Bright's day the determination of chronic albuminuria is considered the best sign of a renal affection; and since Rayer's time many writers have called the affection after him albuminous nephritis—a happy enough name, we must grant, for in the great majority of cases not only is albumin present but it constitutes one of the chief if not the principal symptom of this affection.

The quantity of albumin secreted by the kidneys in parenchymatous disease is usually considerable. Generally it con-

tains from 5 to 8 grammes albumin according to Rendu, from 6 to 12 according to Lecorché; frequently enough the proportion rises to 15 to 20 grammes in the twenty-four hours. Rarely does it exceed this figure; still, Bartels has cited a number of cases where the amount of albumin surpassed 5 per cent.; more rare are the cases where the amount is as low as 1 to 2 grammes to the litre.

Aside from these variations there are others most interesting, which may be noted when account is taken of the daily progress of parenchymatous nephritis. These variations are of two sorts: the first are related to the period of the disease, and the second occur suddenly from day to day; we will dwell a trifle on each variety.

It is an established fact that the quantity of albumin varies during the course of a parenchymatous nephritis. But do these variations coincide constantly with a given period of the disease? An affirmative answer seems to us *à priori* improbable. A cursory glance through the literature of the subject will teach us what slight accord in opinion there is on this point.

Christison [41] first noted that it was during the initial period of the affection that the quantity of albumin reached its maximum; Lecorché [42] accorded with this view. Rayer [43] believes that albumin increases with the progress of the disease. Bartels [44] categorically establishes this; "it is at the acme of the disease, he writes, that this substance is present in greater quantity than at other times."

It should be stated that the majority of writers are in accord in regard to the fact that it is in the last stage of the disease—that of renal atrophy—that the amount of albumin is least considerable; at this stage the decrease may extend even to complete disappearance.

In the same patient, further, albumin may diminish from day to day; these daily variations ordinarily coincide with the variations in the quantity and the specific gravity of the urine. They are in quite constant relation with the density and in inverse relation to the quantity of urine eliminated each day. These changes are not accompanied by any alteration in the health of the patient, and they occur without known cause. An increase or a decrease in the quantity of albumin does not indicate an amelioration or an aggravation of the disease.

This point we must insist upon, for it is not as yet generally recognized.

Absence of Albumin.—"It may happen that the albumin disappears even at the end of the disease, but from time to time, up to death, we see it reappear under the influence of various causes." Jaccoud is still more categorical: "The absence of albumin is even the rule in the last period of the disease." Although the majority of writers are not in accord in regard to the frequency of the disappearance of albumin during the last stage, the greater number admit at least the possibility on the occurrence of renal atrophy; there is far less agreement in regard to the possibility of disappearance during the course of a parenchymatous nephritis.

"Frerichs, Bright, Christison, Malmsten, Johnson, have reported cases where, the albuminuria ceasing, the parenchymatous nephritis still persisted. We cannot *à priori* accept these cases, since albuminuria for us constitutes the pathognomic sign of intra-canalicular inflammation; it may happen, however, that the albumin diminishes, and even disappears."

Jaccoud[45] is less affirmative: "At this stage the albumin may disappear for a few days and then return later."

In case of the large white kidney, then, it seems to us that there may occur transient disappearance of the albumin lasting for some days, or even for some months.

I report the following case, which we met with the current year.

Case I. (*Personal.*)

Bright's Disease (Parenchymatous Form). Transient Disappearance of the Albumin.—M——, æt. forty-seven, was admitted to the Necker Hospital, February 23, 1887, service of Professor Dieulafoy.

The man had had typhoid fever at the age of seventeen; in the month of August, 1886, as the result of catching cold, he had had chills, fever, nausea, bilious retching, pain over the kidneys lasting for two to three days. Soon after there appeared headache, intense dyspnea and nocturnal cramps. These symptoms lasted till October 15; then they became exaggerated and in addition there was swelling of the eyelids, edema of the feet and legs up to the knee, buzzing in the ears, slight deafness, etc. These symptoms caused the man to cease work; he entered the Beaujon Hospital November 10, 1887. While there he suffered from pollykiuria with polyuria (1500

gramme daily), and albumin appeared. On January 19 he felt relieved, left the hospital, and resumed his work.

This condition lasted till the middle of February. Then the chill and fever recurred, as well as the other symptoms. He then entered our service at the Necker.

The dyspnea was then intense, especially at night, numerous bronchial râles were scattered over the lungs, the edema was almost generalized. There was no albumin in the urine. The man was placed on milk diet and a mixture of syr. of ether and of syr. of orange flowers was prescribed.

February 24, urine acid, 1.200 c.c.; no albumin.

February 25, urine alkaline, 800 c.c. no albumin.

February 26, urine acid, clear, 1.750 c.c., no albumin. The dyspnea has diminished but the man feels no better.

February 27, urine slightly acid, sediment, 1.000 c.c., a trace of albumin. The man feels worse; chill; backache, nausea, bilious vomiting.

February 28. Trace of albumin persists; dyspnea exaggerated, numerous râles in the lungs.

March 1, alkaline urine, 400 c.c.; properly tested it yields albumin 14 grammes to the litre. On his body there appeared a vesiculo-pustular eruption; the diagnosis of varioloid was made; at the end of two weeks he was cured, and he was discharged on April 15.

Time and again the examined urine contained albumin. The man returned to work and continued till May 10. He then caught cold, had chills, pain in the loins, the urine became scanty, and micturition was frequent. He remained at home for several days under ordinary diet. The symptoms increased, the edema returned, and he entered the hospital again on May 18, 1887.

May 19, the urine is cloudy, scanty, and contains .75 grammes of albumin (serin).

He remained in the hospital for several months; his urine, examined daily, varied from 1.000 c.c. to 1.600 c.c., and always contained from .25 to .75 gramme albumin (serin).

From June 10 the symptoms disappeared under continuous milk diet; the urine became normal and contained no albumin. Twelve days after he returned home and we have since lost sight of him.

This case is of interest, for it proves clearly that in the course of a parenchymatous nephritis, the albumin may transitorily disappear.

From the 23d to the 27th of February it was absolutely lacking, and then reappeared.

Did this happen only under the influence of the renal affection? Did not the general intercurrent affection have an influence?

During the second stay of our patient in the hospital, we found for twenty-two days albumin in the urine; it then disappeared and there existed no trace during the last twelve days he was with us. Likely enough the treatment caused this disappearance. To resume: in this man, on two different occasions, albumin was lacking; although we can readily explain the second disappearance it is not easy to account for the first.

Do there occur cases where during the whole course of the disease albumin is lacking? We do not believe that this is possible, and we find no such record anywhere. We must except Henoch's case; here albumin was absent during the whole course of the affection, verified post-mortem; it only appeared the evening before death. We give a short analysis of the case:

CASE II. (*Abstract.*)

Scarlatina.—Acute parenchymatous Nephritis; no Albuminuria.—Death.—Autopsy.—A young boy of twelve was admitted into the service for children at the Berlin Charité on July 22, 1873. The face and scrotum were edematous and had been for several days; he was desquamating from scarlet fever of three weeks previously. The urine was red, very acid, no albumin; in the sediment there was present microscopically no element suggesting nephritis.

On the 23d and 24th the edema increased, but there was no change in the urine. The night of the 24th violent attacks of eclampsia occurred, with complete loss of consciousness.

The morning of the 25th delirium and cyanosis; by catheter a small amount of urine was obtained containing a large amount of albumin, and numerous hyalin casts.

The morning of the 26th consciousness returned; persistent cyanosis, dyspnea, imperceptible pulse, dullness at the base of both lungs, fine râles. Death the morning of the 27th.

At autopsy: Parenchymatous nephritis on both sides; serous effusion into peritoneum, pericardium, pleural cavities; edema of lungs, etc.

Such as far as we know is the only recorded case of parenchymatous nephritis without albuminuria—at least, throughout the greater part of its existence. This case we insist, however, has reference to an acute parenchymatous nephritis, and is therefore different from the affection we are now studying.

To resume, then:

Albumin is present in chronic parenchymatous nephritis.

The quantity, generally considerable, varies according to the individual, but in the same patient according to the period of the disease; it varies from day to day, and this too without alteration in the state of the patient.

Albumin may be lacking in chronic parenchymatous nephritis; this is not unusual in the last stage, but may occur in the course of the disease at longer or shorter intervals; never, up to now, however, has a parenchymatous nephritis progressed without albuminuria.

Interstitial Nephritis.—Hyperplasic nephritis (Lecorché); diffuse primitive proliferative nephritis (Lancereaux); renal sclerosis (Jaccoud); gouty kidney; contracted kidney; small red kidney.

Presence of Albumin.—"Even as the urine of parenchymatous nephritis so does that of interstitial contain albumin almost always, but while in the first affection albuminuria is the chief phenomenon and excels all the other phenomena in importance, in the latter albumin is a secondary and contingent phenomenon."

To these words of Rendu, which emphasize the difference in the symptom albuminuria in these two varieties of Bright's, we must add (and this diminishes considerably the value of this symptom in case of the small red kidney) that albuminuria in interstitial nephritis presents marked variations both as regards quantity and progress.

The amount of albumin is ordinarily feeble; it occurs as a slight opalescent cloud, rarely flocculent, appearing slowly and at the end of a few seconds. "This slight proportion of albumin is characteristic of interstitial nephritis, for it is one of the most constant modifications in the urine."[46] A superficial examination may readily not determine it; the urine must hence be examined with great care, and the slightest cloudiness should excite the suspicion of the physician.

According to Wagner[47] the urine of interstitial nephritis does not contain more albumin to the 1000 than that of parenchymatous nephritis does to the 100. The amount that transudes through the renal filter does not usually exceed a few centigrammes, rarely reaching 2 grammes in the twenty-four hours. Bartels[48] gives the following figures: 1 gr. 03, 1 gr. 48, 2 gr. 16. Exceptionally it surpasses these figures, and then at the end of the disease.

Even as in parenchymatous nephritis the albumin varies according to the stage of the disease; it varies frequently enough from day to day.

It is not in the first stage of the disease that albuminuria is established permanently; it develops by starts. From time to time the urine becomes albuminous for a greater or less time; then the albumin disappears to reappear again, and this during months, often indeed during years; it is only at the end of a number of years of this irregular progress that it becomes permanently established.

All writers are here in accord, and the fact has been known for long. Todd[49] first noted it: he had found that frequently albuminuria followed on attacks of gout, and that it totally disappeared in the intervals. Johnson[50] confirmed in full the assertions of Todd. Bartels,[51] Lecorché,[52] Lancereaux,[53] Labadie-Lagrave,[54] Senator,[55] are all unanimous on this point. We may, therefore, conclude with Rendu[56]: "It appears, then, probable that in the first stages of the affection, albumin only appears at intervals, and that the nephritis progresses by starts."

The following case appears to us characteristic in this connection:

CASE III. (*Personal.*)

Bright's Disease (Interstitial Form).—Initial Stage.—D——, æt. sixty-eight, tailor, entered hospital June 27, 1887.

Antecedents good, with the exception of a typhoid fever. For the past three to four months has had the following symptoms: cephalalgia; epistaxis (morning), slight visual and auditory troubles, first phalanx of right hand deadened, nocturnal cramps, electrical starts, frequent nocturnal desire to micturate without polyuria, edema of lower extremities and of thorax.

The urine examined 29th of June to 7th of August, presented the following characteristics:

Slightly acid or neutral; sp. gr. varying between 1.009 and 1.015, the quantity varied between 1.250 c.c. and 2500 c.c., the mean amount being 1675 c.c.

Albumin appeared and disappeared for varying intervals. From 29th June to 3d July inclusive the amount of albumin varied from .35 gr. to .55 gr. From July 4th to July 12th complete absence of albumin. It reappeared on the 13th and persisted to 21st (.35 gr. to .40 gr.). It disappeared July 22 to August 7, and then the patient passed from under our notice.

Absolute milk diet bettered his condition markedly.

English writers are much more affirmative; some of them contend that renal sclerosis, in its first stage, is not accompanied by albumin, and they have called this stage the pre-albuminuric; this opinion, first expressed by Milner Fothergill,[57] has lately been accepted by Mohamed in a number of papers.[58]

Although we cannot admit the pre-albuminuric stage of the English writers, we coincide fully with the French opinion, which seems to us well established: at the beginning of interstitial nephritis albumin appears in the urine after a transient fashion, and this intermittency seems to us the true characteristic of this first period. At the end of a number of months, or of years, when the sclerotic lesions have progressed, the intermittent albuminuria becomes permanent, although it is still subject to variations of which we will speak.

"The quantity of albumin passed in interstitial nephritis may be so great as not to differ at all from that met with in the course of primitive parenchymatous nephritis."[59] Usually, however, it is not pronounced until the latter stages of the disease.

At this period the amount diminishes according to some: "In all cases albumin diminishes towards the end of the disease."[60]

"It diminishes again, and even completely disappears when the vital forces are lowered and the heart weakens."[61]

According to other writers it increases: "In the last stage of life it is not rare to find very abundant flocculi of albumin even as in parenchymatous nephritis; it would then be very difficult to make a differential diagnosis if we did not know the previous march of the disease."[62]

"In a number of analyses made at an advanced stage of the disease I have seen it vary from 10 to 15 grammes.[63]

The albumin, further, is subject in quantity to daily variations.

Gubler,[64] in a paper read before the *Sociéte de biologie* the 6th of August, 1853, describes, in general, these variations in amount, dividing them into periodic, accidental and diurnal variations; he dwelt in particular on the latter. According to him albumin is ordinarily present in greater amount during the daytime; the contrary holds, however, for those who work at night. Bartels[65] has confirmed these views, and has shown

the marked oscillations occurring in the same individual in the course of interstitial nephritis, oscillations, further, in accord with his manner of living, food, and general state of nutrition.

In a number of instances he has noted the alternate disappearance and reappearance of albumin; in one case he checked this for seven months. During this whole period albumin was found in the day urine and no trace in that of the night; when the patient passed his days in bed the albumin disappeared from the urine to reappear when he left his bed for the day.

Wagner[66] has noticed the same fact as Bartels; in certain cases albumin is absent from the night urine, while that of the day, especially when the patient was active, contained a certain amount. Ellis[67] has also noted that the absence of albumin often coincided with rest in bed.

Patients weakened by privation usually have little albumin in the urine when they enter the hospital; as their nutrition increases, the albumin often increases.[68]

Absence of Albumin.—Although it is an established fact that albumin may be absent for a greater or less time in case of renal sclerosis, especially in its early stage, a less established fact, and one less acceptable to pathologists, is the possibility of the non-appearance of albumin during the entire course of the disease.

"The urine, in case of granular kidney, may or may not be albuminous," so wrote Wilkes (1852) in Guy's Hospital Reports.

Since, this question has been discussed by many writers; all have answered it identically, as prove the following quotations; some admit with Bartels that every nephritis must at some time be accompanied by albuminous urine:

"Albuminuria is not a constant phenomenon, as recent observations have proved for me. Füngel mentions that absence of albumin in the urine of his patients has often led him into the error of not recognizing the disease. But neither Füngel's observations nor mine, nor those of others, prove that albumin may be lacking during the entire course of the disease. Albuminuria is, in any event, one of the most characteristic symptoms."

A few pages further on, Bartels[69] cites the only case which he has seen of renal atrophy, verified by autopsy, which, during life, was never accompanied by albuminous urine; and there-

fore, he adds, the existence of the renal disease was not determined.

Many writers, still, are of the contrary opinion; they claim that albumin may be absent in renal sclerosis.

"The urine need not contain albumin; the disease may run its course without there being present the slightest trace; this is when interstitial nephritis has remained uncomplicated by canalicular trouble; usually, however, albumin is present."

Mahomed [70] and Millard [71] are still more positive; according to them interstitial nephritis is not usually accompanied by albuminuria; albumin only appears when epithelial alterations occur.

Between the view of Lecorché and that of Mahomed and Millard there is an intermediate view, that of Professor Dieulafoy.[72] He has proved that there exists Bright's disease without albuminuria, and this he has described in his two papers last year at the Academy of Medicine, and this year in his lectures at the school. We agree perfectly with this view and have often proved the truth of it.

We cite a few cases which prove this point. We give first, in abstract, two cases cited by Dieulafoy in his papers read before the Academy, of peculiar interest in that the diagnosis was confirmed post-mortem.

Case IV. (*Dieulafoy.*)

Bright's Disease (Interstitial Form).—Absence of Albuminuria.—Autopsy.—A woman æt. forty-seven was admitted to the hospital March 19, 1886. Her symptoms were angina pectoris, cephalalgia, frequent desire to micturate and marked sensation of dead finger.

Fifteen days before admission she had a violent attack of angina and there followed edema of the feet and of the legs (March 15). She had, on auscultation, pulmonary edema of the right base; the urine was scanty, high-colored, no albumin. M. Dieulafoy diagnosticated Bright's disease of two years' duration.

March 20, 400 c.c. urine, no albumin.
March 21, 700 c.c.; no albumin.
March 22, 1.500 c.c.; no albumin.
March 23, 2.600 c.c.; no albumin.

The increase in the amount of urine coincided with betterment in general condition as the result of diuretics and milk diet.

The patient caught cold on the 24th; generalized bronchitis

set in and she died on the 25th. The amount of urine fell to 258 gr.; trace of albumin.

At the autopsy the kidneys were of the normal size, and each contained three to four cysts. Histologically, evidence of interstitial nephritis with arterio-sclerosis predominating.

This conclusive case proves clearly that interstitial nephritis may progress without albuminuria; the slight trace of albumin found on the last day does not invalidate this conclusion.

CASE V. (*Dieulafoy.*)

Interstitial Nephritis.—Absence of Albumin.—Autopsy.—This case concerns a patient with Bright's complaining for a considerable time of violent cephalalgia (like that of syphilis), of attacks of oppression, of cramps, absence of sensation in finger, increased desire to micturate, the urine being of mean amount and containing no albumin (6th to 12th of July).

On July 12, bronchitis developed; the urine decreased to a litre, and albumin appeared to the amount of .95 gr. daily up to death.

At autopsy: nephritis with arterio-sclerosis.

We might cite many other cases which we have seen, although, unlike the preceding, we have not the evidence yielded by autopsy. The question seems to us adjudicated in the affirmative. Side by side with the large class of cases of Bright's disease with albuminuria must be ranked the increasing class of Bright's without albuminuria.

We may resume, then, as follows:

The presence of albumin in interstitial nephritis is subject to great variations; ordinarily intermittent at the outset, it usually shows itself when renal sclerosis has progressed.

The absence of albumin during a greater or less period, at the beginning and end of the affection, is frequently noted; it is likely that such may be the case during its entire course. Although we think the English and American views too absolute, we do not question but that there exist cases of Bright's without albuminuria. These cases are few in number to-day, but they will become more frequent. With a deeper knowledge of the many symptoms of Bright's the disease will be better recognized and this too without waiting for the appearance of albumin in the urine—the too common tendency of to-day.

Mixed Diffuse Nephritis.—Between these two extreme types of chronic nephr tis, the one of which is characterized by

the large white kidney with exclusively epithelial alterations, and the other by the small granular kidney with interstitial proliferations, there occurs a mixed type, where sclerosis is mixed with epithelial degeneration, where the inflammation is diffused through all of the renal parenchyma. It is difficult to trace the clinical history of this form, since both the lesions and the symptoms are varied and complex.

Although from the preceding considerations we have been able to clearly prove that albumin is abundant and almost constant in parenchymatous nephritis, and that, on the other hand, it is in small amount and irregular in appearance in interstitial nephritis, it is very difficult to draw the slightest conclusion in this respect in the numerous varieties of mixed nephritis. We might try to classify these nephritides in different secondary groups, basing them on the progress of the affection and on the symptoms peculiar to them; but this division seems to us too schematic, and consequently little in accord with clinical data. Therefore, without emphasizing the diverse combinations which may be met with and the different cases of chronic diffuse inflammation of the kidneys, we will content ourselves with the statement that a close examination readily allows us to say whether the case approximates a parenchymatous or an interstitial nephritis. Having established this point, we may deduce the progress and the characteristics of albuminuria in a given case:

The more the parenchymatous lesions are marked, the more abundant will be the albumin and the more constant its appearance.

The more the interstitial lesions predominate, the less will be the albumin and the more irregular its presence.

According to Wagner,[73] who has been able to follow for a long time a large number of cases in his private practice, diffuse nephritis, which is by far the most common form, and which he thence calls the usual Bright's disease, habitually pursues the following course:

At the outset the albumin is abundant and permanent; then, as the affection progresses, the albumin diminishes, but increases transiently from the slightest cause. Finally, when the terminal stage is reached, and this usually requires two to three years, the albumin not uncommonly disappears.

We give in abstract the following typical case:

Case VI. (*Dieulafoy.*)

Bright's Disease (Diffuse Form).—Absence of Albumin.—Death.—Autopsy.—Woman æt. twenty-nine, entered hospital November 11, 1885. In July, 1884, the symptoms were vomiting and hematemesis; in November the vomiting increased and a second hematemesis. Milk diet kept her well up to May, 1885. The vomiting then returned, accompanied by gastric and lumbar pains and by cephalalgia. In this condition she entered hospital. These symptoms together with the absence of albumin led to diagnosis of gastric ulcer, and she was placed on exclusive milk diet.

Up to December 29th the symptoms persisted; two or three times hematemesis; no albumin. Coma and death on December 29. Autopsy: stomach healthy; kidneys whitish, large, and microscopically evidence of mixed nephritis.

Here then we have a patient who was under observation for seven weeks and never showed the slightest trace of albumin, although she had a mixed nephritis.

We give a second case of Dieulafoy's, which proves conclusively that diffuse nephritis may run its course and albumin may entirely disappear for a longer or shorter time.

Case VII. (*Abstract.*)

Diffuse Bright's.—Disappearance of Albumin for 2½ months.—Autopsy.—This patient, *non compos*, had a trace of albumin on the 14th February, and on the eve of her death, May 4; in the interval no albumin whatever.

The symptoms were uncontrollable vomiting, polyuria, cramps, cephalalgia; edema and albuminuria were alone wanting. Autopsy: diffuse nephritis.

These two cases, with autopsy, confirm the views of Wagner; they suffice amply to establish the point at issue.

Nature of Albuminuria.

For long the existence of albuminuria has been known, and it was further recognized that it existed in many and in varied diseases, and then the question arose as to whether it was possible, at least readily, to distinguish at the bedside cases where the kidney was seriously diseased from those where the presence of albumin in the urine was not dependent on an alteration of the organ. With this end in view the attempt

was made to utilize the properties of the albuminoid substance determined; at the outset its action under various reagents was found out, and next the determination of its nature was aimed at.

To expose the physical characters of the albuminous coagulum, on the one hand, and its chemical characters, on the other, such is the double study we are about entering upon in order to determine the nature of the albuminuria of Bright's disease.

Physical Characteristics of the Albuminous Coagulum.—It is we believe in a brief note to his treatise on the diseases of the kidneys, that Rayer first noted the fact of the retractility of albumin, without, however, attaching the least importance to this property, as is proved by the following extract:

"After having precipitated the albumin in the urine by nitric acid, if an excess of acid be added, in less proportion than the urine, however, the albuminous flocculi almost always become crisp." [74]

Ten years later (1850) this question was studied from a clinical standpoint. In his inaugural thesis Dr. Oppenheimer Seligman [75] writes (a fact already established, however) that there exist two kinds of albuminuria:

The one transient, met with in many diseases;

The other permanent, met with in the granular affection of the kidneys known as Bright's disease.

He adds that in both cases the urine has special characteristics, and that the precipitated albumin behaves in different ways.

In case of transient albuminuria, the urine is high-colored; it has never the lactescent whiteness of the urine of Bright's; the albumin, under the influence of reagents, precipitates with difficulty in flocculi.

In case of permanent albuminuria, connected with Bright's disease, the urine is lactescent, opaline, and almost always whitish; when subjected to reagents the albumin falls quickly in condensed flocculi, snow white or at least clear opaline.

This question of the physical characteristics of the albuminous coagula has been studied again, a few years later, by Professor Bouchard (November, 1880).

In a paper read before the Congress at London on the in-

fectious nephritides, and in two papers read before the Clinical Society, he establishes the following facts which result from the study of many cases of albuminuria.

When coagulated albumin is subjected to the action of heat, it happens that at times the coagulum retracts into flocculi and leaves the urine perfectly clear. On the other hand, a simple homogeneous, lactescent cloud is produced. These two albumin states correspond to two pathogenetic conditions altogether different.

The retractile albumin is that of normal, intact blood, which is able to pass on account of an alteration in the renal filter; this is the albumin which is met with in diseases of the urinary tract, and especially in nephritis.

The non-retractile albumin is an abnormal albumin, capable of dialysis, notwithstanding the integrity of the renal filter; it is found in diseases accompanied by high fever (typhoid, erysipelas, pneumonia, pleurisy, etc.).

This differential property was most important; but, unfortunately, other researches soon invalidated it. Soon after its birth the theory of Bouchard was actively attacked.

Cazeneuve and Lépine, in two papers read before the *Societé Biologique* (November, 1880), showed that retractility or absence of retractility depended not on the intimate nature of albumin, but on the chemical composition of the medium in which the coagulation was affected. If the urine were acid enough the precipitate forms in flocculi; if, on the other hand, it is too acid or not enough so, the urine is clouded, and this changes into flocculi when, according to the case, the liquid is neutralized or sufficiently acidified. Rodet [76] corroborated these views.

The chemical composition, then, of the medium in which the coagulation takes place has a manifest influence on the constitution of the precipitate. Other conditions, which must be taken into account, also have an influence; on the one hand, the quantity of albumin present, and, on the other, the degree of concentration of the urine. These views of Bouchard have been fully confirmed by our colleague, Capitan,[77] in his inaugural thesis; we quote the following passage, which clearly states the case.

"If we examine a normal urine, to which has been added a determined quantity of egg albumin, or a diluted urine of

which we know the amount of albumin present, we will see that proportionately as the quantity of albumin decreases we obtain successively large, medium and small flocculi, and finally the cloud of non-retractile albumin. This in turn ceases to be perceptible."

Let us, for instance, subject to the aceto-picric reagent and to heat different solutions more or less rich in albumin: We note the following facts:

In a sol. 1 gr. to 1000, abundant flocculi of mean size. In a sol. 50 gr. to 1000, abundant, but small flocculi. In a sol. 25 gr. to 1000, very fine, sand-like flocculi. In a sol. 12 to 1000, uniform slight cloud. In a sol. .06 to 1000, doubtful cloudiness. In a sol. .03 to 1000, nothing apparent.

In his different experiments Capitan used different reagents and always determined the following fact:

In proportion as the albumin in a given liquid diminishes, so does the precipitate become less marked; it even ends by disappearing if the amount of albumin is very slight.

These physico-chemical conditions of the albuminous medium invalidate almost completely the clinical value of the manner in which the albumin coagulates. We do not claim, however, like certain writers (Brucke), that the appearance of the coagulum is the worst of all the characters which assist in distinguishing the different varieties of albumin, for in certain cases this appearance may be of great value.

Although, as has clearly proved Professor Jaccoud,[78] the retractile albumin may be met with in typhoid fever, in simple angina, and in pneumonia without there being marked change in the kidney, one fact is certain, and this is that every marked case of nephritis is accompanied by retractile albumin; therefore we may, with great probability of being right, whenever, by whatever method, we obtain a rapid and abundant retractile precipitate, admit the existence of a renal lesion.

May we go further? May we thence deduce the age of the lesion? Jaccoud thinks this possible;[79] witness the following passage which we take from one of his lectures on parenchymatous nephritis:

"I can affirm that the following characteristics only belong to an old nephritis; large flocculi form at once, and they are of a dirty-green color."

We simply cite this fact. It has not been noted by other writers.

Chemical Characteristics of the Albuminous Coagulum.—If the physical characteristics of the obtained albumin have only a limited diagnostic value, does the same hold for the chemical? We must now try to answer this question.

Although thirty years have passed since the discovery of different kinds of albumin in the urine, we are not at one as yet in regard to the value of each species, although some are thoroughly known.

Studied, at the outset, by Mialhe from a purely chemical stand-point, next referred to by certain writers as curiosities, for a long while they were deemed without value. It was only later that the attempt was made to attribute to these different kinds of albumin a significance which we are going to try to expose in stating the question of urinary albumins from the actual stand-point of to-day.

As we have seen, we may find four varieties of albumin in the urine: serin, globulin, peptones, and hemialbuminose or propeptone. We have already exposed their peculiar characteristics, and have already stated the methods of differentiation; we must now determine their clinical value.

Serin.—The albumin of serum or serin is the most important, beyond question, of the urinary albumins; it is, to borrow Jaccoud's happy expression, the albumin of Bright's, *par excellence.* Abundant and constant in parenchymatous nephritis, rare and intermittent in interstitial nephritis, it exists in all cases of Bright's disease, at least in those which are accompanied by albuminuria. The same remarks hold true for serin as did for this symptom, and we need not dwell on it further.

We are content then with simply mentioning serin, and we pass at once to the clinical history, far less known, of the other urinary albumins, globulin, peptones, and propeptones.

Globulin.—Lehmann,[80] one of the first to call attention to this albuminoid substance, found it in nephritic urine; according to him it is present with serin, although in less proportion. Edfelser[81] grants the view of Lehmann, but established the fact that the quantity of globulin was in direct proportion to that of serin. Such is not, however, the opinion of Gerhardt,[82] who, while admitting the existence of globulin, claims that it is not found in the urine of all cases of Bright's. Fuhry-Suethlage[83] and Leube[84] have also detected it, the first in a case of diffuse nephritis, the second in a case of parenchymatous nephritis.

Petri[85] has made numerous analyses; in fourteen cases of chronic nephritis he only found globulin four times. It is in particular Senator,[86] of whose competence in regard to albuminuria every one testifies, who especially studied this question (1874-1878). Not satisfied with finding globulin in albuminous urines, he queried if we might judge the character of the renal affection by the greater or less quantity of globulin. With this end in view he determined the quantity of this substance present in the different forms of nephritis; he found that it was particularly abundant in case of the amyloid kidney, and in acute nephritis; he also found it in chronic nephritis; rarely in diffuse nephritis; directly in proportion to the amount of serin in circumscribed nephritis; therefore Senator concluded that in chronic nephritis the amount of globulin met with has a true diagnostic value, for it allows us readily enough to differentiate the different varieties.

The views of Senator are far from being accepted without protest; not alone in Germany, but also in England and in France, this question is far from being accepted.

In Germany, Heynsius[87] claims that the presence of globulin furnishes absolutely no information in regard to the nature of the disease.

Hoffmann[88] established by numerous analyses that the connection existing between the amount of globulin and of serin may change notably in the same patient; consequently a diagnosis based on the relations existing between these two albuminoid substances may lead to marked error.

In England, Laundby[89] is not less affirmative; in an important work which appeared in 1880, he concludes that globulin exists rarely enough in the urine, and always concomitantly with serum albumin; consequently it cannot as yet answer as a characteristic of a morbid group.

The same views are current in France; it suffices to quote the following passages which we borrow from two clinicians who have especially studied the subject of albuminuria:

"The valuation of globulin in the urine furnishes no presumption in regard to the nature of the renal affection."[90]

"It is impossible to establish any connection between the nature of the renal lesion and the presence of globulin in albuminous urine. In consequence globulin proves nothing in regard to the existence of a renal lesion; if this lesion is not

made probable by other symptoms it tells us nothing either as to the nature or the degree of the lesion; it possesses then no diagnostic value whatever in regard to the state of the kidneys; it is purely an index of a non-determined modification of the condition of the blood."[91]

A few pages further on Jaccoud, after having insisted on the importance clinically of separating these two urinary albumins, claims that globulin exists in false albuminuria, and in the transient albuminuria which occurs during the progress of or as a sequel to acute diseases, and which depends on the modification of the proteid substances of the blood by the acute disease. In this connection he refers to a patient with acute pneumonia; the urine subjected to the ordinary reagents gave a precipitate; deprived, according to rule, of globulin by the sulphate of magnesium, it gave no further precipitate by any reagent; in this case of pneumonia the urine was totally lacking in serin and contained only globulin.

This fact is very interesting; we need other similar cases in order to accept the ingenious opinion of Jaccoud. Quite recently certain papers have appeared on this subject.

The most complete and most conscientious is without question that of Dr. Kamensky,[92] published at St. Petersburg in 1885. After an analysis of the question, he states the results of his researches; he determines with great care the daily amounts of globulin and of serin; he next establishes the difference between them; he next compares the relation existing between the total amount of albumin and the serin. Of the four observations of this writer one alone allows of conclusions, and we give it in abstract.

Case VIII. (*Kamensky.*)

Chronic Diffuse Nephritis.—B.——, baker, æt. 31, alcoholic and syphilitic. In the spring of 1884, symptoms first appeared. Swelling of the face in the morning, edema of the legs at night, occasionally violent cephalalgia.

Patient entered hospital 19th September, 1884; abundant flocculi of albumin in the urine.

The first exact analysis was made the 27th of December, the last the 18th March, 1885. Here are the figures:

Date.	Quantity of Urine.	Percentage.	Total Weight Albumin.	Weight of Serin.	Weight of Globulin.	Relation to Amt. of Albumin.
Dec. 27, 1884.	1,700 gr.	.77	13.22 gr.	9.65 gr.	3.57 gr.	2.70
Jan. 9, 1885.	2,500 "	1.02	24.60 "	14.86 "	9.80 "	1.61
" 19, "	2,730 "	.96	26.24 "	18.85 "	7.39 "	2.55
Feb. 25, "	1,830 "	1.17	21.48 "	14.24 "	7.24 "	1.95
" 28, "	2,400 "	.63	15.34 "	10.60 "	4.74 "	2.33
Mar. 1, "	2,800 "	.42	11.83 "	8.90 "	2.93 "	3.44
" 18, "	2,200 "	.57	12.53 "	9.44 "	3.12 "	3.02

If we examine this table we may determine, generally, the following facts:

We cannot establish any comparison between the amount of albumin and fractions of albumin.

We may deduce the fact that the relation of the albumin is not constant; generally, it diminishes as the serin increases, and it increases as the latter diminishes.

An examination of the three other observations does not allow the same deduction. Therefore Kamensky concludes that in a given case there does not exist any relation between the amount of albumin and that of globulin, and that the latter, hence, has little diagnostic value.

As for us we regret that in this interesting study the writer did not take into account the relation existing between the amount of albumin and the general state; and he might possibly have drawn some striking conclusions. This point he has not at all considered, nor have any other writers on the subject.

After Kamensky's work it remains for us to notice that of Chéron,[93] which appeared last year in *l'Union médicale;* in his review of the clinical value of the varieties of urinary albumin our colleague accepts entirely the ideas of Jaccoud. He insists on the importance of differentiating these two urinary albumins, which are precipitated by the same regents, because:

"Globulin does not indicate a renal lesion when it exists alone.

"When it coexists with serin the amount of the latter is alone important in diagnosis."

In the experiments we had occasion to make for a greater or less time on twelve patients, we found globulin only in four; this urinary albumin, usually in small amount, was found on different occasions for two or three days; it appeared and disappeared without our being able to note anything special in the condition of our patients. We must mention, however, that in the last patient in whom we found it, the appearance of the globulin and its increase have coincided with the appearance of a violent uremic headache, the prelude of sharp attacks of eclampsia, in the midst of which our patient died.

We here report the four observations of globulinuria which we have met with:

Case IX. (*Personal.*)

F——, æt. twenty-four, entered the service of Professor Dieulafoy, Hospital Necker, June 21, 1887.

This woman, at the time of her first pregnancy (two and a half years previously), had urinary disturbances, among others frequent desire to micturate. A few months after delivery, she complained of sharp pains in the right lumbar region, soon followed by persistent oppression and cough. A physician from the *Hôtel-Dieu*, who then saw her (March, 1885), diagnosticated bronchitis and placed her on milk diet, under which *régime* she improved rapidly.

One month later the same symptoms appeared and in addition swelling of the eyelids and marked edema of the legs; she again went to the *Hôtel-Dieu* and was again placed on milk diet.

She improved, but two months later the symptoms became aggravated and she was admitted to the *Hôtel-Dieu*. Absolute milk diet again caused disappearance of symptoms.

From August 2, 1885, to November 15, 1886, the patient remained well, symptoms being held in check by occasional milk diet.

From November 15, 1886, her health deteriorated; nausea and at times uncontrollable vomiting was added to the dyspnea and the edema; next there followed nocturnal cramps, recurring about every eight days, epistaxis, slight, in the morning, urinary disturbances. All other symptoms of Bright's were lacking.

In this condition the woman entered the hospital. Under milk diet she rapidly improved, and on the 3d of July she seemed in perfect health.

The examination of the urine made daily from the 22d of June to the 15th of July showed:

The urine, usually slightly acid, had a specific gravity of 1008 to 1010.

The quantity varied from 3000 c.c. to 1500 c.c.; the mean quantity being 2290 c.c.

The amount of albumin varied from 9.25 gr. (July 6th) to 0 gr. (July 15th) the mean daily quantity being 6.30 gr.

At various times we noted the presence of globulin:

June 22d, 2.50 gr.; 24th, 25th, 28th, 30th of June and the 1st of July, only traces.

On the 10th of July it appeared as a flocculent cloud floating in the liquid; she then had headache and slight visual disturbances. The globulin persisted to the same degree the 11th to the 14th inclusive, the cephalalgia disappeared on the 13th, as also the visual troubles.

On the 15th the globulin reached the amount of 2 gr. to the litre, or 4.50 gr. total quantity; the liquid cleared of the abundant precipitate yielded by the sulphate of magnesium contained no trace of albumin.

On this day F—— left the hospital. We have never seen her again, and we regret this, for the case was of great interest. Such as it is it proves that globulin, in its evolution, follows an intermittent course, and that it appears or disappears without appreciable cause.

Case X. (*Personal.*)

C——, æt. fifty-one, entered hospital May 17, 1887, service of M. Rigal. In perfect health up to the end of 1885, without known cause he was seized by shortness of breath on climbing the stairs, and by edema of the legs and eyelids. Soon thereafter appeared frequent nocturnal micturition, cramps, dumbness of phalanges of left hand.

He entered Necker and his urine was found loaded with albumin; at the end of a month he was discharged cured, without albumin.

For a year to a year and a half his health was perfect and he kept at his work. Early in May, 1887, the symptoms returned and he again entered the hospital. The legs were edematous as well as the face and the thorax; the amount of urine was normal, but it contained albumin in marked amount; no other symptoms of Bright's. He was placed on milk diet and dry cups to loins were ordered daily.

The urine examined daily from the 17th of June to July 10th showed us the following:

Urine feebly acid; specific gravity varying from 1008 to 1018.

The amount varied from 800 c.c. to 2000 c.c.; the mean amount being 1110 c.c.

The quantity of albumin varied from 1.4 gr. to 4 gr.; the mean daily quantity being 2.5 gr.

The globulin appeared, irregularly, on several occasions:

On June 10th, 19th, 20th, about .50 gr.; the 21st, 22d June only traces. On 29th and 30th June it again appeared as a slight flocculent cloud, and again on the 3d and 4th July; finally on July 6th and 8th the cloud was a trifle more marked.

During this interval there was nothing specially marked in the symptoms; from June 28th all symptoms of Bright's disappeared. Since then the patient has been in good health.

This observation is especially of interest in that it concerns a case of Bright's without albuminuria.

CASE XI. (*Personal.*)

Bright's Disease.—(Interstitial Form.)—Absence of Albumin; Intermittent Globulinuria.—R——, æt. sixty, entered hospital June 17, 1887, with paresis of right lower limb, which had followed a fall from the third story in July, 1886. He complained further of shortness of breath and frequent desire to urinate. He had also edema of the legs and eyelids, cramps in the calf, epistaxis, a few drops, in the morning. He had, further, a few visual disturbances and deafness of both sides; *bruit de galop* quite marked.

Under milk diet he improved rapidly, the edema and most of the other symptoms disappearing in a few days. The shortness of breath persisted to the end of July.

The urine from the 29th of June to August 12th presented the following characters:

Slightly acid, alkaline or neutral; specific gravity varying from 1005 to 1016.

Maximum amount 4000 gr. July 6th, minimum amount 1100 June 29th; mean daily amount 2910 c.c. At no time albumin. Globulin appeared on three separate occasions, always in scant amount; on the first occasion July 2d, on the second July 6th to 17th, on the third August 3d (quite distinct cloud).

This observation of a Bright's patient without albuminuria is the only instance where we have found globulin. Dr. Estell of Lyons, in his thesis, reports two cases where globulinuria alone existed, the first concerning an atheromatous patient, the second an ataxic.

CASE XII. (*Personal.*)

Bright's Disease, Diffuse Form.—Albuminuria, Globulinuria, regular in its course, Eclampsia, Death.—G——, æt. twenty-five, entered the service of Professor Dieulafoy

November 22, 1887, for edema of limbs and dyspnea. Two and a half years ago the patient caught cold and then suffered from violent cephalalgia and anasarca; the urine was scanty, not bloody, and contained flocculi of albumin; he had been placed on milk diet and followed this for three weeks. Since that time he has only complained of occasional cephalalgia and of frequent micturition without excess in amount.

From September, 1887, his cephalalgia became more frequent and intense; at times he was nauseated and had bilious vomiting; often edema of the legs; sometimes a little swelling of the eyelids in the morning. He caught cold November 14, 1887; suffered from slight fever, cough and hoarseness.

On entrance into hospital he had laryngo-tracheitis and slight bronchitis, frequent, distressing cough, abundant gurgling, sub-crepitant râles scattered over the lateral thorax. On examining the urine we found flocculi of albumin and he was placed on milk diet.

November 24, urine slightly acid, 2400 c.c., 6 gr. albumin (only serin).

November 25, urine acid, 2300 c.c., 5¾ gr. albumin.

November 26, urine acid, 2350 c.c., 5 c.c., 20 albumin.

November 27, slightly acid, 2300 c.c, 4¼ gr. albumin.

The laryngo-tracheitis has gone and patient feels well.

November 28, urine acid, 2200 c.c., albumin 5.20 gr.

November 29, slightly acid, 1800 c.c., 2¼ gr. albumin.

November 30, 1600 c.c., slightly acid, 2.40 gr. albumin, slight cephalalgia.

December 1, increase of the cephalalgia; urine very alkaline, lactescent, charged with urates, 1500 c.c.; 2¼ gr. albumin; first appearance of globulin (sulphate of magnesium test).

December 2, headache persists; urine slightly acid, clearer, 1500 c.c., 2.60 gr. albumin, globulin manifest.

December 3, more headache, insomnia, urine 1300 c.c., 2.60 gr. albumin; flocculi of globulin.

Treatment: 1 leech to nucha; sinapisms.

December 4, continuous headache; insomnia, nausea; urine alkaline, 1300 c.c., 1.65 gr. albumin, globulin.

December 5 symptoms worse; urine still alkaline, 1600 c.c., 1 gr. serin, .60 gr. globulin. No urine during the day, the bladder empty, by catheter.

December 6, at 5 o'clock A.M., first attack of eclampsia. 6 leeches behind the ears.

At 5¾, second attack; at 6¾, third; at 8, fourth; at 9, fifth; at 9¾, sixth; temperature 37.3°, venesection to extent of 800 gr.; inhalations of oxygen.

At 10½, seventh attack; at 11½, eighth. At noon, return of consciousness, and desire for sleep.

At 10½, evening, ninth attack; death.

Autopsy 36 hours P.M.: lesions of diffuse nephritis.

Before we can give a personal opinion we need more cases; for the time being we are obliged to rest content with the narration of our observations. We have only proved that globulin does not exist in all cases of chronic nephritis; when it does exist it seems to follow a rather irregular march, appearing and disappearing without appreciable cause.

Peptones.—We have not determined as much in regard to peptones. On many occasions we have hunted for them in our albuminuric cases, both by the direct method which seems to us quite imperfect, and also by precipitation by the acetic solution of phosphotungstate of soda; we have never found them. We have also often examined from this stand-point the urine of cases of Bright's without albuminuria, and we have never obtained the reaction of peptones.

Are our methods of research imperfect? We have always used the methods which are recommended. Must we admit that peptones are rarely found in albuminous urine? This we cannot answer.

This question of peptones is indeed much in dispute; according to some, albuminuria and peptonuria are often associated, and according to others they are never.

We are here naturally led to sketch briefly the history of our third species of urinary albuminuria.

Described by Mialhe[94] under the name of albmuinuria, studied by Gerhardt[95] under the name of latent albuminuria, the peptones were especially studied from a clinical stand-point in Germany and in Italy.

In Germany peptones have been found in the urine of Bright's by a number of observers, Eichwald,[96] Obermuller[97] and especially Senator,[98] who has detected them in all febrile and afebrile conditions characterized by the presence of coagulable albumin in the urine; Pétri[99] found them nine times in fourteen of chronic nephritis. Maixner[100] and especially von Jacksch[101] have thoroughly studied this question; the latter studied 354 cases, which include 762 urinary analyses, but we do not find mention of renal affections.

In Italy writers are not so affirmative in regard to the association of albuminuria and of peptonuria. Pietro Grocco[102] of Paris considers peptonuria as utterly independent of albuminuria; it is found especially in case of local inflammatory and suppurative processes, rarely in chronic affections.

This interesting question of peptonuria has been studied in France from a different stand-point; its connection in renal disease has attracted little attention.

"Peptonuria may appear indifferently in all cases of Bright's; it presents nothing in accord either with the nature of the lesion nor with the existence of a lesion; it depends on the state of the blood and not on that of the kidneys." [103]

It seems then that if peptones exist in albuminuria their presence has no diagnostic import.

8. *Hemialbuminose.*—This little known agent has been found in renal affections; Fuhrbinger and von Jacksch have found it, the former twice, the latter once, without drawing any deduction. Lépine thinks that its presence points to a dyscrasia worth nothing.

Connection between Albuminuria and the Urinary Secretion.

Now that we know the information which the existence and the nature of albuminoid substances in the urine may give us, we must seek if there do not exist relations between albuminuria and the urinary secretion, and if it be not possible to draw from such relations certain considerations useful from the diagnostic point of view of Bright's disease.

We will consider as before parenchymatous nephritis, interstitial nephritis, and diffuse nephritis; the two former in particular since their characteristics are more marked, although the diffuse form is the most frequent. We must pass successively in review the physical characters, the chemical characters, and the microscopic characters of the urinary secretion in chronic nephritis.

Physical Characters of the Urine of Bright's—Although in the majority of cases the urine of Bright's presents certain external characteristics which at first sight differentiate it from normal urine, we must bear in mind the fact that these characters are of secondary importance and are not of much assistance in establishing a diagnosis.

Although it is true that the urine is yellow, deep, more or less cloudy in parenchymatous nephritis, and pale, transparent, opalescent in interstitial nephritis, there are many modifications to these statements. Further, we must not forget

that, at the outset, the urine of chronic nephritis is like normal urine.

The same remarks hold true for the reaction and the specific gravity. In short, there exists, among the external characteristics of the urine, only one which we think is of importance: this is the quantity of urine passed, and on this point we will dwell somewhat. We wish first to insist upon the fact that all the urine passed each day must be measured.

Parenchymatous Nephritis.—Although we do not know much about the quantitative modifications which occur in the first stages of the large white kidney, we know that at this time the amount of urine secreted is usually below the normal; all writers are here in accord except Frerichs,[104] who contends that at this period the amount is increased; probably this observer, as have pointed out Lecorché and Rendu, has confounded primitive parenchymatous nephritis with certain forms where the lesions of the large white kidney are associated with lesions of renal sclerosis.

The urine rarely exceeds 1300 to 1500 grammes; it generally oscillates between 900 and 1000 gr. (Rendu), 500 to 600 gr. (Charcot), 350 to 800 gr. (Lécorche), 698 gr. (Bartels)—such is the mean amount he has deduced from 122 observations; at any rate the amount remains much below the normal, as long as the inflammation is of the same degree of intensity (Bartels).

An important point is that the amount varies greatly from day to day; the amount of drink taken, the method of treatment, the period of the disease, the intercurrent affections, these are factors which modify more or less the amount of urine passed; it appears difficult then to lay down any hard and fast rule (Rendu).

In advanced stages of the disease the amount may attain the proportions indicated by Frerichs. This increase is also noted when the nephritis tends towards cure. "As soon as the nephritis runs a favorable course the quantity of urine increases." (Bartels.)

Interstitial Nephritis.—From the start, the quantity of urine is increased; polyuria exists, particularly at night, and this fact attracts the attention of the patients, so that we are able to determine quite readily the beginning of the disease. This polyuria varies, however, with the patient. In some the amount does not exceed 2 litres; in others it attains 3 to 4 litres and even 5 to 6 litres (Bartels).

The polyuria, further, is not constant in its progress, often indeed we see it replaced by a true dysuria. One fact is established, and this is that in the later stages of renal sclerosis, when the heart begins to fail, whatever the cause, the amount of urine diminishes notably, in some instances as low as 180 to 200 gr. daily; in other cases a short time before death there occurs complete anuria, usually followed by uremic accidents; after the cessation of these accidents it is not unusual to see the amount of urine increase.[105]

Diffuse Nephritis.—In the numerous intermediate varieties, where the parenchymatous lesions are mingled with the interstitial, the course of the urinary secretion is very variable. According to Wagner the urine is at the outset diminished in amount; little by little, as the color of the urine becomes paler, the amount increases until at the end of a few months it passes the normal. Still, from time to time, under the influence of various causes (catching cold, for instance), the amount diminishes coincidently with an inflammatory or a congestive attack. These changes in the amount of urine in diffuse nephritis progress besides with the albuminuria; scanty when the albumin is abundant, the amount of urine increases as the albumin diminishes. It is not always so, however; in our eleventh case, which appears to be one of diffuse nephritis, we note the figure for the urinary secretion and that for the albumin diminish concomitantly.

Such are the various external characteristics of the urine in Bright's disease, can we draw any deductions from the side of diagnosis?

Professor Lépine, of Lyons, at the Congress of the *Association française pour l'avancement des Sciences*, held at Grenoble in August, 1885, laid down certain fixed rules in regard to the value of the symptom albuminuria; he tried to prove that, although in many cases a persistent albuminuria is a symptom of ill-omen, it is yet insufficient alone to allow us to affirm the existence of a serious lesion of the kidney; in other cases, however, in connection with many other symptoms it becomes a valuable sign of Bright's disease. According to the Lyons professor here are certain of the conditions:

Albuminuria, he says, is a positive sign of chronic nephritis, in case it appears in one of the following conditions:

In large amount:

The percentage increasing concomitantly with the polyuria:

Medium percentage, but total quantity exceeding 8 gr. (owing to the coexisting polyuria):

Small percentage, but persisting for a long time.

A few months afterwards one of his pupils, in his inaugural thesis, endeavored to prove the correctness of these observations. Manin, indeed, reported some conclusive facts in his article *"De l'albuminurie comme signe du mal de Bright."* It seems to follow clearly from this study that when albuminuria is present under certain conditions specified by the professor at Lyon it may be considered as a true index of the disease.

Of these four conditions the first and the third are frequently enough met with; the two others more rarely, but they are still important, particularly the second; this characteristic, indeed, according to Lépine, allows us to differentiate Bright's albuminuria from cardiac where the percentage of albumin diminishes as the quantity increases. Too frequently, however, these two forms of albuminuria are present together, and this is apt to invalidate our research.

For our part, we have as yet been unable to verify all the assertions of the Lyons school; but we are inclined to admit the first and the fourth conditions, which are often clinically true.

Chemical Characteristics of the Urine of Bright's.—Do the chemical properties of the urine of chronic Bright's give us analogous information? In certain cases may they give us true indices of the affection? These points we proceed to study.

The modifications in the chemical composition of the urine of Bright's have for long excited attention; since it was established that albuminous urine was not the exclusive property of renal affections, and that it was found in numerous affections, local and general, the attempt was made to find out if the albumin of Bright's did not accord with certain peculiar changes in the composition of the urine. English physicians, in particular Christison and Gregory, declared that albuminous urine had a low specific gravity and was poor in salts and in urea. French physicians tried to verify these assertions, and searched for the different modifications of the urine in chronic nephritis, aside from the presence of albumin. Rayer insisted especially

on the lowering of the figure for urea in albuminous nephritis; according to him, any dropsy accompanied by albuminous urine and diminished density, and especially diminution in urea, indicated with almost absolute certainty a renal affection. The opinions of Rayer were granted by most clinicians who studied this question; it became quite generally admitted that diminution in urea, diminution in salts, lowering of the density, were three factors which marched hand in hand with albuminuria in Bright's disease. To-day, the chemical composition of Bright's urine has almost entirely lost its significance; it is very difficult to deduce any clinical indications from the chemical composition of the urine.

Without stopping to examine in detail the changes which occur in the different substances (extractives and minerals) in the urine of Bright's (for in the present state of our knowledge such an examination has not much value towards diagnosis), we will only consider the modifications of the most important of the urinary constituents after albumin, and this is urea. It will be clearly apparent after we have studied it in the two opposed forms of Bright's, large white kidney and small red kidney, how restricted is its value from a diagnostic standpoint.

In parenchymatous nephritis the amount of urea is in direct proportion to the specific gravity of the urine; this is the richer in urea the less the quantity in which it is secreted. Such at least follows from the observations of Bartels.

At the beginning of the affection the quantity of urea is notably diminished, and this increases with the progress of the affection; the figure is always above the normal, oscillating usually between 12 and 15 gr., rarely exceeding 20 gr. (Charcot). Still, and this must be emphasized, the quantity of urea varies not only in individuals, but in the same patient both proportionately and absolutely (Bartels). It is evident, as justly states Labadie-Legrave, that in the Bright's patient with edema and abundant dropsical effusion the elimination of urea by the urine will be less active, since this product of disintegration escapes in part through the vessels with the effusions into the skin and the serous cavities; the same holds true for the subject to Bright's whose nutrition is compromised by abundant loss of albumin; in the latter, indeed, the proportion of urea and in consequence its elimination are restricted within appreciable limits.

If we compare these clinical facts with Lehmann's experiments it is easy to understand of what little importance is the quantity of urea excreted in parenchymatous nephritis. This observer has, indeed, proved that normally, under the influence of a simple modification in the diet, the quantity of urea may triplicate. How then take into account the variations of the urea in this affection? Likely enough the kidney plays a *rôle* in the small proportion eliminated, but the *rôle* is certainly a very secondary one.

Does the same hold true for interstitial nephritis? The study of the variations of urea in this form of Bright's is not more instructive; it is less, if this be possible.

For a long time the urea remains at its physiological height; the patient excretes from 25 to 30 gr. in the twenty-four hours (Bartels), a fact which is worth remembering. It is only later that the amount decreases to such a degree that we at times only find 1 to 3 gr.[106]

Under these conditions it is readily understood that daily dosage is of little value, and furnishes but few elements useful for diagnosis. This daily search, however, is not a useless one; if it does not assist in diagnosis it still furnishes the physician useful indications in regard to the nutrition of his patients.

Microscopic Characteristics of the Urine of Bright's.—If the chemical composition of albuminous urine has not much value from a diagnostic stand-point, the microscopic examination of the urinary sediment has not much more.

For a long enough time writers have attached great importance to the study of the urinary sediments, and especially of the casts; persuaded that they represented exactly the lesions of which the kidneys were the seat, they endeavored to distinguish carefully the different varieties, and they described in great detail the hyaline casts, the granular casts, the granular-fatty casts, the waxy casts, etc., each variety belonging to a determined renal lesion. Soon, however, it was ascertained that these casts were often found without the kidneys being much affected, so that they lost entirely in value. To-day they are hunted for rather as curiosities than as elements in the diagnosis. Still we must dwell on them a trifle, since, although they may be found in cases where the kidney is not affected, they are always found in case of chronic nephritis, and we may by their characteristics determine the variety of nephritis which is present.

In parenchymatous nephritis the urine is usually cloudy; the sediment is abundant and under the microscope are found crystals of uric acid, urates and phosphates, leucocytes, sometimes blood corpuscles, casts of varying kind and number. Bartels, who has made a careful study of casts, says: "The majority are pale, hyalin, or slightly striated, and often carry *débris* of epithelial cells. The more chronic the case the more frequent the granular casts, the larger they become and the more frequent the waxy casts."

In diffuse nephritis there is little sediment, and it contains fat cells and a few casts; not uncommonly there is no sediment for days in succession.

Casts are sparse in interstitial nephritis; microscopically a few crystals of urate and of oxalate of soda are found, and a few hyalin casts, exceptionally some granular. According to Robin[107] there is also found a notable quantity of urohematin. In short, the microscopic examination allows us to determine certain differences between the varieties of Bright's disease; but it is not necessary to hunt for casts in order to specify the variety of nephritis. To-day, therefore, the microscopic test is of restricted value.

Connection between Albuminuria and the General Condition.

Is it possible to deduce from the relations existing between albuminuria and the general condition data of utility from the stand-point of diagnosis of Bright's disease? This question we propose studying from a clinical stand-point.

In parenchymatous nephritis, together with the albumin which is present as a rule, we must note the edema which, in this affection, pursues a special course. At first transient and localized in the morning on the eyelids, at night at the malleoli, it soon becomes permanent and generalized over the body; its progress is peculiar enough to suggest the large white kidney even before the albumin appears to confirm the suspicion.

Although the diagnosis of this variety of chronic nephritis is usually easy, certainly, in the majority of cases, the same is not always true of renal sclerosis; the scarcely pronounced albuminuria, often absent, the slight dropsy, attract scarcely the attention of the physician when certain cephalic, digestive,

respiratory troubles lead into error. This he may often avoid by consulting the heart. The hypertrophy of this organ, so well described by Bright, is suggested to the ear by the *bruit de galop*, the characters of which have been well established by Professor Potain; but this valuable sign may be lacking and the diagnosis remain uncertain. Often still, in quite a number of cases, the attention of the clinician is attracted by special visual signs; a progressive amblyopia due to a retinitis of quite marked characteristics, leads to the thought of the possibility of an interstitial nephritis; therefore, in doubtful cases, an ophthalmoscopic examination should not be neglected. By never neglecting the data furnished by the heart and the fundus of the eye the diagnosis of renal sclerosis may often be established in its initial stage.

Although the special progress of edema allows us to suspect parenchymatous nephritis, and the heart murmur and the peculiar retinitis point to interstitial nephritis, affections which the albuminous urine in many instances will confirm, how much more difficult is it to make a diagnosis in all those obscure forms of Bright's so frequently met with, which are characterized, some by persistent headache, others by diverse sensorial troubles, others by dyspeptic trouble, others by a greater or less difficulty in breathing, others still by deep anemia, etc. If in these varied instances, so different the one from the other, the urine contains albumin, the diagnosis is, in general, easy; but if it be not present, how difficult the diagnosis! The diagnosis must be made, however (and this is essential both to the physician and the patient from the stand-point of treatment), it must be made, we repeat, and we must have the courage to order the rigid treatment required by such a diagnosis; cure, or, at most, rapid amelioration of the disease will soon justify the diagnosis.

To make the diagnosis in these difficult cases we must search with care for those little symptoms which our teacher has grouped under the generic name of lesser accidents of Bright's. cephalalgia; sensorial disturbances (buzzings, diminished vision, whistlings, more or less marked deafness, epistaxis in the morning); disturbances of the general sensibility; cramps; muscular twitchings; digestive troubles; respiratory troubles; urinary troubles.

All these accidents, which we can only mention, have, true

enough, only slight value when isolated; together they should lead to the right goal and should allow us to reach the diagnosis of Bright's even when careful repeated examination of the urine shows absolutely nothing abnormal.

We see, then, that it is not useless to study the general condition; careful search often gives valuable aid, not alone to complete the diagnosis, but in many cases to establish it with certainty.

Actual Diagnostic Value of the Symptom Albuminuria in Bright's Disease.

Having reached the end of the first part of our study it seems to us appropriate to resume our conclusions:

Albuminuria, abundant in the course of parenchymatous nephritis, is met with nearly constantly in this affection.

It is, on the contrary, rare in interstitial nephritis, in the course of which it occurs intermittently and is subject to great variations; in certain cases, even, infrequent enough to-day, but which will certainly soon become more frequent, albumin is entirely absent.

In the many varieties of diffuse nephritis its characteristics and progress vary greatly; we cannot, therefore, specify its principal traits.

In Bright's urine we do not always find a single and similar albuminoid substance; there are different varieties. One is met with constantly (serin), the others with greater or less frequency according to the case (globulin, peptones, propeptones).

At present we cannot infer anything to speak of from their presence or their absence; they neither specify the nephritides nor do they differentiate the clinical varieties.

The chemical composition of chronic nephritic urine has not the importance formerly granted it. The same is true of the characters furnished by the microscopic examination of the sediment, notably the casts.

The physical characters alone of the urine, although of less value than formerly, deserve recognition. The quantity of urine passed in the twenty-four hours not only allows us to differentiate the two chief varieties of nephritis, but it also

permits under conditions well specified by Lépine to separate the renal affection from those which may simulate it.

The general condition of the subject of Bright's must always be taken into account; it almost always furnishes useful information. The information indeed is often sufficient to allow us to affirm Bright's disease even when albumin is absent.

Finally, albuminuria has certainly a diagnostic value, but it is restricted enough when taken separately; its value is wider when considered in connection with the urinary secretion and the general state of the patient; we are then in a position, often, to affirm the renal lesion and to specify the variety.

BIBLIOGRAPHY.

1, De ischide nervosa commentarius, Vienne, 1770.—2, Case of Diabetes Mellitus, London, 1798.—3, Trans of Soc. for the Improv. of Med. and Chir. Knowledge, vol. iii., 1812.—4, Obs. on Nature and Cure of Dropsies, London, 1883.—5, Edinb. Med. and Surg. J., vol. xxx., 1827.—6, Reports of Medical cases.—7, Diseases of the heart, of the large vessels, of the liver, serous inflammations.—8, Bright's Work, p. 75, et seq.—9. Edin. Med. and Surg. J., vol. xxxii., 1829.—10, Ibid., vols. xxxvi. and xxxvii. 1831.—11, De l'Albuminurie, etc., 1838.—12, Dissertatio de quodam vitis, quod urinæ mutatio particularis comitatur.--13, Th. Paris, 1833.—14, Lettres à Rayer sur l'Albuminurie, Gaz. Med., Paris, 1837.—15, Clin. Med. Charité, 1837.—16, Vol. ii., 1840.—17, Clin. Lect. on Dropsy, 1830.—18, Lond. Med. Gaz., 1835,—19, Ibid.—20, Guy's Hosp. Reports, 1836, 1840.—21, Th. Agreg, 1857.—22, Virchow's Handb. der spec. path. u. ther. vol. vi., 1865.—23, Anl. des Harns, 1878.—24, Virchow's Archiv., vol. xxxii., 1878.—25, Dict. Jaccoud, art. Urine, p. 452.—26, Brit. Med. J., Nov., 1878.—27, Guy's Hosp. Rep., xxiii., 1872.—28, Rev. Med. 1882.—29, Albuminuries transitoires, Th. Paris, 1883.—30, Recherches sur l'Albuminurie physiologique, Th. Paris, 1883.—31, Berl. Klin. Woch., 1885.—32, Ibid., 1886.—33, Ibid.—34, De la maladie de Bright, Th. Paris, 1884.—35, Albuminurie Symptome dans les Maladies, Th. Paris, 1859.—36, Union Medicale.—37, Tr. Mal., Reins, p. 392.—38, Brit. Med. J., vol. ii., 1879.—39, Chronic Bright's Disease without Albuminuria.—40, Nephrites sans albuminuries, Th. Paris, 1884.—41, On Granular Degeneration of the Kidneys, and its connection with Dropsy, 1839.—42, Loc. cit., p. 208.—43, Loc. cit., p. 115.—44, Loc. cit., p. 354.—45, Loc. cit., p. 419.—46, Loc. cit., p. 120.—47, Rendu: loc. cit., p. 121.—48, Labadie-Lagrave: Tr. mal. des Reins, 1888.—49, Loc. cit., p. 438.—50, London Med. Gaz., 1845.—51, Med. Chirurg. Trans., 1850.—52, Loc. cit.—53, Loc. cit.—54, Dict. Dechambre.—55, Mal. Reins, p. 783.—56, Archiv. f. Path. Anat. u. Phys., vol. xliii., p. 1.—57, Loc. cit.—58, Brit. Med. J., 1878.—59, Guy's Hosp. Rep., vol. xxv., p. 295, 1881.—60, Lecorché: loc. cit.—61, Ibid.—62, Bartels: loc. cit.—63, Rendu: loc. cit.

—64, Lancereaux: loc. cit.—65, Dict. Dechambre, p. 507.—66, Loc. cit. —67, Loc. cit.—68, Boston Med. and Surg. J. 1880.—69, Bartels: loc. cit. —70, Loc. cit., p. 436.—71, Loc. cit.—72, Loc. cit.—73, Loc. cit. and Gaz. Hebd., 1886.—74, Labadie-Lagrave, p. 760.—75, Loc. cit., p. 114.—76, Albuminurie comme symptôme dans les maladies, Th. Paris, 1850.—77, Lyon, med., 1882.—78, Th. p. 1883.—79, Clin. med. Char., 1885.—80, Loc. cit.—81, Virchow's Archiv., vol. lvi.—82, Deutsch. Arch. f. Klin. Med., vol. vii.—83, Prag. Virch. f. pract. heilk, 1871.—84, Deutsch. Arch. f. Klin. Med., vol. xvii.—85, Zeit. der Phys. Med. Loc. Zu Erlangen, 1878. —86, Dissert. inaug., Berlin, 1876.—87, Deutsch. Arch. f. Klin. Med., 1878. —88, Ibid., vol. xxii.—89, Virchow's Archiv., vol. lxxxiv.—90, Brit. Med. J., 1880.—91, Rendu: loc. cit., p. 97.—92, Jaccoud: loc. cit., p. 567.—93, Vratch, 23.—94, De la valeur clinique des diverses varietés d'Albumine dans l'Urine, 1886.—95, Acad. X., 1846.—96, Prag. Viert. f. pract. heilk., 1871.—97, Deutsch Arch.—98, Beitr. zur chem. der Eiweis, Wurzburg, 1873.—99, Die albuminurie, Berlin, 1881.—100, Prag. Viert. f. pract. heilk., 1879.—101, Prag. Med. Woch., 1881.—102, Ibid.—103, Ann. Univ. di Med. e di Chirg., Napoli, 1883, 1884.—104, Die Brightische nierenerkrankung, Braunschweig, 1851.—105, Wagner: In Labadie Lagrave.—106, Dickinson: Pathology and Treat. Albuminuria, 1868.—107, Dict. Jaccoud.

CHAPTER III.

THE PROGNOSTIC VALUE OF THE SYMPTOM ALBUMINURIA IN BRIGHT'S DISEASE.

At the outset of the first part of our study we deemed it useful to recall the phases through which the question of albuminuria has passed, in the belief that these historical considerations would enable us to rapidly arrive at the value of this symptom in the diagnosis of Bright's disease. To follow the same course here would be to lose time; indeed writers on this subject have paid but little attention to this point. In reading their monographs, although we find the prognosis of chronic nephritis well exposed, we meet with but few details in regard to the *rôle* which the symptom albuminuria plays in prognosis.

To take account of this *rôle* we must again study different factors: the existence and nature of albuminuria on the one hand, the connection between the urinary secretion and the general health on the other hand; in fact the same factors, the same examination, although more briefly.

Existence of Albuminuria.

The irregular progress, the frequent absence, the small usual quantity of albumin in renal sclerosis makes us think at once that in this variety of Bright's it would be very rash to attach much value to this symptom. Whence why all writers on this subject—Rayer, Charcot, Rendu, Rosenstein, Lancereaux, Bartels, etc., have not paid much attention to this point.

Lecorché, alone, suggests the prognostic importance of the presence of albumin in the course of interstitial nephritis. "When," he says, "the albumin is abundant (and this may happen at any stage of the affection) this is a sign that parenchymatous nephritis complicates the affection; the prognosis

becomes grave, for the loss of albumin is a further cause of weakness which soon leads to a condition of anemia."

Almost constant, notwithstanding its many variations, in parenchymatous nephritis, the albumin deserves serious consideration.

In general the presence of albumin in the urine has been always looked upon as a phenomenon of ill-omen; for the majority of clinicians, indeed, when the urine becomes albuminous the prognosis becomes very grave; as Bartels says, "if heat coagulates the urine in a test tube the patient is condemned."

Many, however, think that the unfavorable prognosis attributed to the symptom we are studying has been much exaggerated.

"Albuminuria is a bird of ill-omen in diseases of the kidney, to which an extreme importance is attached, behind which we expect the edemas and all the precocious or tardy symptoms of Bright's disease, and which are accused of provoking weakness. Still it is often a few centigrammes, at most a few grammes of albumin which the patient passes each day; such a slight spoliation is not capable of deteriorating the health. A nurse loses without harm 40 to 50 grammes of albumin or of proteid matter through the function of lactation and yet her health is not deteriorated." *

Is it really true that albumin must always retain its bad reputation? Or must we admit with Bouchard that it has been wrongfully accused?

To answer this question two points are, we think, to be considered: the quantity and the course of albuminuria.

Christison thinks that the greater or less amount of albumin cannot assist in prognosis. "The chances of death," he writes, "are not at all proportionate to the quantity of albumin in the urine. The reverse is true to a degree, for when albumin is abundant the organic derangement of the structure of the kidneys is in its first stage."

Rayer is not at all of this opinion, and looks upon abundance of albumin as an unsavory phenomenon; such at least is the conclusion to which his numerous observations have led him.

Since, the majority of writers have considered the abundant

* Bouchard: Auto-Intoxication dans les Maladies, 1887.

loss of albumin as grave; but have attached a greater or less importance to it.

"The prognosis is serious when persistently the urine contains a large amount of albumin, for every circumstance which increases the organic loss at the expense of the vital resistance constitutes but another unfavorable condition." (Rendu.)

"If the renal affection is accompanied by fever or by other phenomenon which interferes with nutrition and repair, the great albuminuria becomes a cause of weakness, even as does an abundant leucorrhœa, dysentery, suppuration, frequently evacuated ascites. An abundant albuminuria may cause impoverishment of the blood." (Bouchard.)

The abundance of albumin is, therefore, a point to be considered; it furnishes, indeed, certain useful information.

Much more interesting, however, is that furnished by the progress of albuminuria in parenchymatous nephritis.

During the evolution of this affection, on which we have dwelt at length, occur two kinds of variation, which are far from being of the same importance.

The first which occur from day to day appear without known cause; more or less frequent according to the case, they are not accompanied by any change in the health of the patient. They must be recognized, however, since for long it was thought that an increase or diminution in the albumin was regularly in accord with aggravation or amelioration of the other symptoms. In looking over the cases of Rayer we find a number of quite marked cases. This is far from being the rule; therefore, in general, to base a prognosis, of one sort or another on the increase or the diminution of excreted albumin, would be *à priori* to run the risk of great error.

But although these daily variations have not the same importance that were formerly attributed to them, the same does not hold true of the changes occurring regularly from time to time. These are generally consecutive to treatment. We will dwell on them for awhile.

When a subject to Bright's is placed on milk diet a number of things may happen:

Sometimes the albumin, after having progressively diminished from day to day, disappears entirely from the urine. This disappearance is complete and certain when, the patient having been placed on ordinary or mixed diet, the albumin

does not return; the prognosis here then is eminently favorable. It is less so when the suppression of the milk and the return to ordinary diet is followed by a sensible and persistent increase in the amount of albumin. Truly, while in the first instance we may look upon the patient as completely cured, we must, in the second, consider him as having still a nephritis in the stage of evolution and we should prepare for the accidents which occur so frequently in the course of parenchymatous nephritis.

Again, after having progressively diminished for a greater or less time, the albumin remains stationary. The patient has 1 to 2 grammes of albumin, oftener only a few centigrammes, or even only traces. What should be the prognosis in such a case, so frequently met with?

Although many clinicians claim that we ought not to admit a cure of the disease until the albumin has entirely disappeared, there are others who believe that we need not attach to the presence of this substance such great prognostic significance; as a proof of what we say we will quote the following passages from two writers who have made a special study of chronic nephritis:

"While albumin remains in the urine, even in small amount, even though all the other symptoms have disappeared, we must not fall into error, for the property of this disease is to present in its course the most marked oscillations and it is not rare to see the albumin increase at the same time that the other symptoms reappear." (Lecorché.)

"If," says Jaccoud, "suppression of milk diet and drugs and return to ordinary diet, are not followed by any variation in the albumin, and this for a number of consecutive months, the patient may be considered cured, notwithstanding the persistence for an indefinite time of a few centigrammes or of traces of albumin. Without question this patient is more subject to nephritis than another, and should take special precautions against cold, dampness and errors in diet, as also abuse of alcohol and exercise; but with regard for these rules he retains his health notwithstanding the presence of this insignificant amount of albumin. I have seen after the lapse of 3, 4, and 5 years patients thus cured up to a few centigrammes, and I have found that they retained their health notwithstanding this remnant of albumin."

Finally, at times, the amount of albumin does not vary or varies but little as the result of treatment and remains considerable. The prognosis is grave, not because a large amount of albumin is excreted daily, but because this amount indicates that the kidneys are markedly affected; and we know from the researches of Brucke that it is the extent rather than the depth of the lesions which renders parenchymatous nephritis grave.

A consideration, then, of the existence, quantity and variations of albumin gives, in case of the large white kidney and of mixed nephritis, useful information. It is not, however, sufficient to allow us to base on these data alone the prognosis of the disease. We must join other factors, to which we now pass.

Connection of Albuminuria with the General State.

In case of Bright's we must never neglect to inquire into the general health. The latter, in general, behaves as does the albumin, becoming worse as it increases, and better as it diminishes. There are, however, certain exceptions worth noting:

Often we see, notwithstanding a marked diminution in the albumin, the general health remains bad; in the presence of these facts we are forced to admit that the disease runs its course; the prognosis should be greatly reserved. Certain symptoms may, however, persist without aggravating the prognosis; thus it is not rare to find in pale and anemic subjects a persistence of dropsy after complete disappearance of dropsy. We may still believe in complete cure of the affection and give a prognosis accordingly. (Lecorché.)

Again, and the cases are frequent, the patient seems to have recovered his health and the albumin persists; we have sufficiently insisted on this point.

We see then that albuminuria and the general state are subordinate the one to the other, and that we must always consider them together if we wish to establish a certain prognosis.

Connection of Albuminuria with the Urinary Secretion.

The relations existing between albuminuria and the urinary

secretion also play a certain *rôle* from the stand-point of prognosis which it is of importance to recognize. For the sake of completeness we must pass successively in review the physical, chemical and microscopical characters of the urinary secretion.

Physical Characters.—Rayer has chiefly studied this question, and we borrow most of our details from his work.

From a physical stand-point two characters of the urine are to be considered: the quantity of urine and the specific weight.

The quantity of urine passed is certainly important to consider; but it is difficult to trust the deductions thence drawn. Notwithstanding every precaution the patients often lose a part of their urine. If, however, we can measure the exact amount we can consider to advantage the progress of the secretion.

An increase in the amount of urine in the twenty-four hours, aside from the drink taken, is a good sign, especially when this increase coexists with diminution of the dropsy and the albumin. This increase may, however, be transient, and may be soon followed by a more or less marked arrest in the secretion, which is an index of increase in the symptoms.

Diminution in the amount frequently foretells an increase in the existing dropsy or the appearance of dropsy.

When the diminution is more marked, and, of course, when there is complete suppression, the prognosis is very grave; for this indicates the approach of cerebral symptoms.

The specific gravity is also of value, for it furnishes valuable information.

When the density increases, and this is due to an increase in the salts, especially of urea and the extractives, the prognosis is very favorable. This, however, rarely happens.

When the density diminishes, the sign is a bad one if the amount of urine secreted is not increased, and if the amount of albumin remains stationary

Microscopical Characters.—To these characters much consequence was formerly attached, but to-day they do not possess much importance. We will not dwell on them.

The Chemical Characters.—Urea has for long played the chief *rôle* in the chemical constituents of the urine.

"A notable diminution in the urea is a bad sign, whether it co-exists with an abundant albuminuria or not; for in these cases death has frequently followed quickly on cerebral accidents." (Rayer.)

Lecorché and Rendu are not less categorical:

"The diminution in urea is a sign of such value that it answers for making a very grave prognosis if the albumin has also diminished."

"The danger is all the greater if the urea is eliminated in great abundance and the alimentation remains stationary."

The important *rôle* granted urea has been much exaggerated: "For long considered as the great poison, urea is not specially toxic." (Bouchard.) He shows, indeed, "that solutions of urea only cause death when injected in the strength of more than 122 c.c. to the kilogramme of the animal, a dose at which pure water kills."

The toxic agent is not the urea, but the total amount of the excrementitious substances contained in the urine.

Professor Jaccoud has held the same view for twenty years. In his lectures at the *Charité* in 1867 he showed that uremia was not due to the accumulation of urea in the blood, but of the materials of the urine. The urine is then toxic, not because it contains a greater or less quantity of urea, or of other element, but it is toxic in totality.

In view of the toxic power of urine it was of the highest importance to find out the nature of the toxic agent in Bright's disease, since, as our teacher says, "in this affection the point of ill-omen is not what happens, but what does not happen."

What matter if the patient passes a greater or less quantity of albumin or of urea? What is of importance is to know how the diseased kidney functionates; what, in other terms, is its degree of permeability. Does the kidney in twenty-four hours eliminate the amount of poison which the organism manufactures in twenty-four hours? This is the question to be answered before we can make our prognosis.

This question is readily answered by seeking in a given case the degree of toxicity of the urine.

Professor Bouchard has shown in his experiments that the amount of poison necessary to kill a kilogramme of a living being, the urotoxia as he calls it, is on an average 50 c.c. of urine, the mixture of twenty-four hours. This fact having been established after the most positive manner, we can readily take account of the urinary toxicity, and in consequence judge the state of the kidney.

Basing his researches on the discovery of Bouchard's, our

teacher has determined time and again the toxicity of the urine of Bright's. We reproduce the following determinations, which seem to us conclusive enough:

Case XIII. (*Abstract.*)

Bright's Disease.—Absence of Albumin.—Feeble Toxicity of the Urine.—Patient æt. forty entered hospital for lumbar and abdominal pains; for some time she has had sensation of "dead finger," cramps and pollakiuria. The diagnosis of Bright's was made by Dieulafoy.

The quantity of urine varied from 1200 to 1400 c.c., and it was poor in urea and in salts of potass; it contained no albumin.

A rabbit of 2 kilo. was taken; 80 gr. injected caused dyspnea and tremor; 90 gr. myosis; 150 gr., salivation, exophthalmia, convulsions; 125 gr. abolition of ocular reflex; 130 gr. convulsive movements of the masticators, and of the neck, respiration almost abolished; 160 gr. death.

The rabbit should have been killed by 100 gr. of normal urine; 160 gr. killed it; the urine of this patient was less toxic than normal urine.

Case XIV. (*Abstract.*)

Bright's Disease.—Absence of Albumin.—Very slight Toxicity of the Urine.—Patient æt. forty-two, admitted May 8th, violent cephalalgia.

Her symptoms dated back four years when, without appreciable cause, edema of the feet and legs, headache, lumbar pains, cramps, and dyspnea, set in. At the end of three months this group of symptoms had about disappeared. At the end of two years cephalalgia, visual and auditory symptoms; precordial pains, lumbar pains, set in. The urine was about normal in quantity, poor in urea and in salts of potass; for twenty-six consecutive days no albumin.

Rabbit of 2 kilo. taken; 230 gr. injected caused dyspnea, myosis and prostration; at 260 gr. the rabbit could not hold up its head, in ten minutes it had diarrhea and abundant micturition; it shook its head, walked a trifle, and in a half hour was itself again.

This animal should have been killed by 100 gr. normal urine.

Case XV. (*Abstract.*)

Bright's Disease.—Absence of Albumin.—Feeble Toxicity of Urine.—Entered hospital for intense cephalalgia, which had lasted for a number of years. Two years previously had had anasarca, dyspnea and whistlings in ears. Three months

ago there had appeared cramps, pollakiuria, "dead finger," etc.

No albumin in urine. Into a rabbit of 1995 gr. 97 gr. urine was injected without symptom.

285 gr. was necessary to kill a rabbit of 2.210 kilo.

We would add further that the urine from non albuminuric Bright's (*vide* our observation No. 5) was also non-toxic; 260 gr. were needed to kil[illegible] bit of 2700 gr.—that is to say, exactly double the quantit[illegible] of urine.

From these experiments there follows the following fact: generally the urine of patients supposed to be Brightic, and yet non-albuminuric, is less toxic than normal urine.

Similar experiments were made by our teacher in case of Bright's with albuminuria, but we cannot as yet state the deductions.

The above experimental method is of great interest; if at times it may be very useful from the stand-point of diagnosis, it is always useful from that of prognosis, since it allows us to judge quickly as to whether the urinary depuration occurs or not.

Such experiments, however, we must emphasize, prove but one point: this is, that on the day or days when made the urine was or was not toxic. To go further than this would be, it seems to us, to fall into great error. To state positive conclusions it is necessary to examine the urine in question every day. There are disadvantages in this, however; in the first place such experiments take time, and in the second place they are quite expensive.

The discovery of Bouchard's, however, is most important. In order to reach a prognosis we must, in particular, base our opinion on the toxicity of the urine.

If the urine is not toxic the elimination is incomplete: the kidney does not functionate, it is certainly more or less altered in structure.

If the urine is normally toxic, the urinary depuration is complete: the kidney functionates regularly; the affection, if not cured, is at least much ameliorated

Nature of Albuminuria.

There is one matter which so far we have left in the shade, and this is the question of urinary albumins. This question is

in dispute. The diagnostic importance is little known, and their prognostic is less known. We only know that serin has pathological importance; the other albumins are only the indices of a non-determined modification of the blood. (Jaccoud, Lépine.) They furnish absolutely no information from the stand-point of prognosis.

We believe, however, that when we have become familiar with the search for these albumins, and when we have carefully noted the conditions under which they appear, we may draw useful deductions; we may then be able to follow quite precisely the progress of the renal lesion.

Actual Prognostic Value of the Symptom Albuminuria in Bright's Disease.

We draw the following conclusions:

For a long time, in order to reach a prognosis in Bright's disease, the existence, the progress, the variations of albuminuria were taken into account, as well as its connection with the general health and the condition of the urinary secretion.

These characters allow us, true enough, in certain cases to reach a prognosis, especially when the *ensemble* is considered; they are, however, insufficient to enable us to judge the question definitively.

We know, to-day, that the prognosis depends on the permeability of the kidneys; we must know in order to reach our prognosis, whether the kidneys allow or not of the complete elimination of the toxic matters manufactured by the organism.

This search must be made repeatedly to enable us to reach any conclusion.

Later on, perhaps, we may be able to judge the question more readily by taking into account the nature of the excreted albumin. If this supposition is confirmed, and it seems to us likely, we will have at our disposal a more practical method than the daily examination of the toxicity of the urine.

CHAPTER IV.

CONCLUSIONS.—ACTUAL CLINICAL VALUE OF ALBUMINURIA IN BRIGHT'S DISEASE.

HAVING passed in review in as detailed a manner as possible the various factors which may give us information in regard to the nature of albuminuria, we believe we are in a position to answer the question we proposed for solution. We may, indeed, actually fix the clinical value of the symptom albuminuria in Bright's disease.

We need recall here only the salient conclusions which may be deduced from the study.

The clinical value of the symptom albuminuria in Bright's, whether considered from the stand-point of diagnosis or of prognosis, is rather restricted; in order to affirm the renal affection, in order to state its progress and consequences, albuminuria may answer, but not alone.

Whatever the case, it should be recognized, for it furnishes valuable information.

If in many cases albuminuria, by its progress and characteristics, enables us certainly to recognize and to affirm Bright's disease, and often indeed to state the variety, there are other cases, also numerous, where its absence or its inconstancy would lead us into error, if we could not depend on information drawn from the urinary secretion and the general condition. Albuminuria, then, has a certain diagnostic value, but too often restricted if considered alone.

Its prognostic value is much less; to depend on it alone is altogether insufficient; to the information it gives us must also be joined that furnished by the urinary toxicity, which for the present must remain the great prognostic criterium.

To conclude: In Bright's disease, albuminuria has but a secondary value, at least in a large proportion of cases.

NEURASTHENIA

AND

ITS TREATMENT.

BY

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NEURASTHENIA AND ITS TREATMENT.

GENTLEMEN:

The diseases of the nervous system, and in the front rank neurasthenia and diseases of the mind, constitute the pathological signature of the intellectual era in which we live. Statistics have proven the rapid increase of the psychoses, and the multiplication of insane asylums proves the truth of such data. In regard to neurasthenia statistical data as to frequency are not obtainable; but the experience of physicians, in particular those who make a specialty of nervous diseases, teaches that it is a condition very frequently met with. The cause of this we find in the rapid intellectual development of our day. The immense progress in the line of intellectual, political, and social development cannot take place without an unusual expense of intellectual elasticity, and the maintenance of and increase in these acquisitions demand a continual tension of the intellectual and psychical forces of all brain-workers. The more complete and many-sided the accomplishments of the statesman, the teacher, the artisan, etc., the greater the demands which arise for the fulfillment and completion of his task, and the greater the mean requirements on the nerve force of the highest in the nation.

This is not all, however. In accordance with the demands on the working force of individuals, so do the requirements of life increase, and not alone as regards the coarser necessities, but also in regard to the luxuries which find their expression in the satisfaction of ambition, in the education and rearing of children, etc. The sufficiency of humble life, the contentment in the fulfillment of duty within one's vocation, the aim to make the inclination and education of children run in the

same groove and towards the same goal as was sufficient for the father, such things are rarely met with nowadays. Every one desires to go higher, not alone for his own sake but also for that of posterity; no one is satisfied with his lot. Whence strife, and political, financial, and industrial crises. Every one hurries in the struggle for existence and for life, and in the daily routine of the large cities this is especially marked. Only in the smaller cities does the observer note the quiet doing of daily work. There people are seen to go slowly; there there is time for rest, for recreation.

When to the restless life and struggle of the large cities other unusual conditions are superadded, when the regular daily tension of individuals is magnified by heavy home burdens, when sickness in the family, anxiety about food, grief over disobedient children, family dissensions, etc., come as an extra burden, then the most elastic nature yields to the weight. Experience, however, proves that even the most intelligent minds, although they seem to be lucky in the upward struggle, will eventually suffer harm as regards their capability from the intensity and restlessness of their work, owing to the lack of recreation and the ill-suited kind of refreshment and food which they take. As the work increases so does the profit; when rest and indulgence are needed by the brain, then is enjoyment and recreation bought at the expense of bodily struggle. Who attempts to do too much in a given time must sip his life enjoyment in a concentrated form. The result then naturally is not repose, strengthening and refreshment of the overworked nervous system, but is fatigue in another direction, and this in turn leads to weariness and to strain.

Nowhere are the results of this struggle for existence and search after enjoyment and profit so marked as in the United States of North America. It was there that the disease-term neurasthenia saw the light and was baptized by Beard. It is noteworthy that Beard himself calls it an American disease which is absent from no household where the inmates use their brains. It is met with certainly in all other intellectual States, but by no means with the same enormous frequency as in the United States. To those who are familiar with the social conditions there this is not surprising. The intensity and haste of the daily work, the restlessness during the time given to enjoyment, the short and broken sleep, the party and domes-

tic strife, etc., peculiarly stamp the social life of the United States.

I have often had the pleasure of giving advice to these worked-out American neurasthenics, and I might cite a large number of examples. Two types must, however, suffice.

An old merchant at the head of a large establishment in New York suffered for years from sleeplessness, pressure in the head, etc. He stated his daily life to have been as follows: I work constantly from eight in the morning to ten at night. I have no time for eating; I usually do so standing, and frequently my food is cold and uninviting before I can take it. At ten at night I am so worn out that I have scarcely the strength left to close my books. During the night my daily work repeats itself in my brain, so that it is usually only towards morning that I obtain a few hours of restless sleep. In the morning I am therefore still worn out, and I am obliged to resort to brandy in order to put myself in shape for work.

A young merchant, who for months has been unable to do any mental work and passes sleepless nights, thus sketches the manner of life he has led for years: We work from eight to six. We have one quarter of an hour's time for lunch. At night, when the day's work is done, we young fellows go to a café, and there we eat and drink and are merry up to two or three o'clock in the morning. I naturally obtain but little sleep, since I must be up and doing by seven in the morning. In case I am obliged to travel on business I do so at night in order to have the day for work. Can you wonder, gentlemen, that such an ill-treated brain finally refuses to work? Where is Kant's golden rule which gives to man out of the twenty-four hours, eight for work, eight for recreation, and eight for sleep? In case of employees who, when their work is done, need not trouble their brain more, this rule may still be observed. Here, of course, there is no neurasthenia.

In addition to the injury suffered from a too close application to business, from domestic troubles, from financial stress, the nervous system is also severely strained by abuse of alcohol, of morphine, and by sexual excess. Of the spirits it is particularly the concentrated alcohols which weaken the nervous system, which depress brain function and in the end totally ruin the organism. True enough the intellectual force may be stimulated by the use of alcohol, even urged to marked

exertion. In time, however, the reaction of the brain is lowered more and more by the use of alcohol, and it depends eventually on external conditions, in particular on the quality of the alcohol, how long the organism can exist. Many can stand it for long, others soon yield.

The action of morphine is still worse, seeing that tolerance leads quickly to increase of the dose, and the injections must ever be repeated at shorter intervals in order to quiet the nervous system and make it capable of work. The neurasthenia of the opium-eater is all the more painful, seeing that he knows full well the ill effects of the drug and yet he cannot resist the craving, and so he lives in conflict with his own knowledge.

Of special pathological importance is the sexual excess of the young during the period of development, above all masturbation and its consequences, impotence, spermatorrhœa, etc. Excessive copulation, as in the newly married, rarely alone leads to a weakness of the nerve force, if other causes are absent. Very frequent, on the other hand, is loss of intellectual and of psychical force in the onanist. He is no longer able to sustain mental work, his power of thought is clearly impaired, his courage is gone, his energy is shattered. These results are especially injurious at the time when the brain and body are developing, for then, in consequence of the onanist's inability to use his brain, a portion of his education is neglected. Further, he suffers constantly under the moral effect of his habit with the knowledge that he alone is to blame for it, and also from the manifold hypochondriacal symptoms which result from this abuse of life and of health. His self-accusations are increased much by the fear of nervous diseases, etc., which may be dispelled by the physician, but in consequence of which the onanist resorts to all sorts of quackery and cures which only land him deeper in the mire.

An important factor, further, in the development of neurasthenia is an hereditary neuropathic disposition, a congenital tendency to weakness and to exhaustion; this property can lie in the family or it may be the peculiarity for generations back, or both factors may be at work.

This disposition may come from a neurasthenic father or be inherited from the mother. It is ordinarily evidenced in childhood by a great impressionability to nervous disturbances,

but it breaks out first with all its phenomena about twenty, when the brain and the nervous system have acquired their full development. Or else, in case of good external conditions, it remains latent until an excessive trouble or severe nervous disturbance causes the eruption of the diseased condition. Often here diseased conditions are at work which, were the individual not hereditarily predisposed, would not harm him. The intractability of the affection to treatment and the quick recurrence after apparent cure, even under good external surroundings, are typical of hereditary neurasthenia.

Race and ancestral peculiarities play a large *rôle* in the etiology of neurasthenia. The Semitic race is especially predisposed to neurasthenia. A neurotic strain, indeed, runs through the entire race, notwithstanding the strength and toughness at its command. Obstacles and difficulties the Jew overcomes with astonishing energy; but a nervous depression, sickness in the family, overcome him entirely. The effect which a sad occurrence, such as the death of a member of his family, has upon his impressionable brain is a very deep and lasting one. Similarly the failure of a financial speculation, the loss of property, may at once awaken neurasthenia with full force. Connected with this, however, there is not necessarily present inability to work; the neurasthenia is here especially psychical, showing itself in depression of spirits.

The picture in case of the Sclavonic and Oriental races is different. In the Russians neurasthenia, especially among the higher classes, is innate. In no nation do we find so many prematurely used-up brains, so many prematurely old people; this has not its origin in a too early, or too great indulgence in life's pleasures, but it is based on a primary exhaustion of the nervous system which leads prematurely to a partial or total incapability of the intellectual powers.

The great intellectual productivity and freshness which our fathers granted to a healthy body, to a healthy mind and to a life full of rich work, are less frequently found in Russia than elsewhere.

If now, after having reviewed the most important etiological causes of neurasthenia, we seek for a definition of the origin of this many-sided affection, we at once meet obstacles, since the anatomical changes at the bottom of the affection are not yet known. We must accordingly rest satisfied with referring

to the pathological causes as functional or as nutrient and irritative. These words mean that, in view of the multiplicity and mutability of the disturbances and the absence of evident organic disorder, we have only to deal with the cure of readily accessible disturbances. Indeed the recuperative power and the change in symptoms is characteristic, although we frequently enough meet cases where the deep effects and frequent recurrence render the affection difficult if not impossible to cure.

We must, then, rest satisfied with a definition from a purely clinical stand-point. I would, therefore, define neurasthenia as a functional weakness of the nervous system, varying from the slightest degree in single localities to entire loss of strength in the whole nervous system. This deficit in strength shows itself at times in the functions of the centres of intelligence, of psyche, at times more in the reflex and restraining centres, and finally more along the course of the nerves. Hence why there are disturbances along all the nerves, and so it is apparent why the clinical picture is a most changeable one, Proteus-like, and not alone in the degree to which the single disturbances develop, but also in the combination of different disturbances and in the kaleidoscopic change of the manifestations.

Nevertheless it is possible to differentiate certain elemental forms of neurasthenia, as Beard has done in his work, namely, a cerebral form, or cerebrasthenia; a spinal form, or myelasthenia, and a universal form, where the entire nervous system is subject to the disturbances.

It is not possible as yet to sharply differentiate neurasthenic disturbances from the other universal neuroses, especially hysteria and hypochondriasis, and from many forms of psychoses, in particular melancholia and mania. We must remember that neurasthenia and hysteria are as frequently combined as neurasthenia and hypochondriasis, so that many writers consider them the same. As to the connection with melancholia, mania; and hallucinatory disturbances, experience proves that they may directly and silently develop from neurasthenia.

The differentiation of spinal neurasthenia from other affections of the cord, such as tabes, myelitis, meningo-myelitis, is less uncertain. These and other organic diseases of the cord are only rarely mistaken for neurasthenia.

The clinical picture of neurasthenia changes, as I have stated, greatly according to the seat and the quality of the essential disturbance. We must, in order to work correctly, differentiate as many forms of neurasthenia as there are organs in man's body, since the majority of these may be the seat of neurasthenic disturbances. In consequence a number of divisions have been made, as neurasthenia of the heart, of the stomach, of the intestines, sexual neurasthenia, etc. We see no utility in such subdivisions. It will be sufficient if I make you acquainted with the most essential phenomena and if I bring those points in particular into the foreground which are of especial importance for the diagnosis.

The cerebral disturbances lead all the others, not because they occur more frequently, but because they interfere with the capability of the individual to the greatest degree. The most essential of these cerebral symptoms are the following:

Inability to do Mental Work.—Here also we meet with many variations; intellectual impotence in all directions, or only in the direction in which the thoughts of the worker lie, as, for instance, inability to count in an accountant, or to preach in a preacher. The lack of ability may be variably great. At the outset it may only consist in a lack of staying power in intellectual work. The brain of a cerebral neurasthenic may still remain highly productive, since originality, acuteness of thought and ready conception may be fully present with the lighter degrees of wear of the nerve force. Beard has aptly compared such brains to a battery of small elements with small potential force and small internal resistance, which momentarily develops an enormous actual force, but quickly becomes inconstant and is soon useless.

In such cases the lack of working force is only too often stimulated by resort to drugs, and alcohol and morphine play a great *rôle* in these patients. They are conscious ever of their brain weakness and depression of spirits, and it is only natural that they should endeavor to assist these symptoms by resort to drugs, in order to be able to attend to their work. That the primary disturbance cannot thus be checked by the addition of new injurious factors, that in this way the tired brain is only more deeply injured, is self-evident. The fact is, indeed, that in the United States neurasthenia goes hand in

hand with alcoholism, morphinism, cocainism and other offsprings of social pathology.

Ordinarily a variety of other symptoms are superadded to this cerebral neurasthenia, which are very characteristic; in the front rank is head pressure, the sensation of a permanent pressure on the head increased at every use of the intellectual powers, next pain and compression in the occipital region and the neck, sensitiveness of the eyes to light, sleeplessness, psychic irritability and peevishness. The nervous asthenopia renders it difficult for the individual to read, while again the quick exhaustion of the nervous force makes it impossible for him to use his brain otherwise. In this pitiful condition, although ordinarily there through their own fault, are the many sexual neurasthenics, the onanists who from their habit of life are driven to books in order to study them, to examine themselves, and yet after a short while they are obliged to cease in their research, for the reason that they do not intelligently grasp what they read. Then they may take long walks, to come home again and read with the same result.

Next come the parasthesiæ in the head, the sensation as if a hoop was around it, as if water was shaken up and down in it, as if the upper eyelids were pulled down. Further, a sensation of fullness in the face, sensation of pricking and burning or coldness of the extremities, lack of sensation in the limbs, tinnitus aurium, and other subjective acute noises, attacks of cardiac palpitation, vaso-motor disturbances, such as pulsation in the epigastrium, the pulsation in the ear and head which can be felt and heard when the ear lies against the pillow, sudden sensations of cooling or of pricking heat, momentary contractions in certain groups of muscles, as though an electric current were passed through them.

The various sensations of fear and of oppression from which these individuals suffer are also characteristic. At times these may be traced to frightful experiences, but usually no cause can be determined for the sensations. The different forms under which these feelings of oppression appear have led to various terms applied to each as if each were a disease *sui generis.* The most comprehensive of these terms is agoraphobia, the fear of large spaces or of broad streets which the patients cannot cross alone, while, if they have the arm of an attendant they can do so readily, although not without a

sense of restraint. This phenomenon, which manifests itself in such varying manner, is not due to a fear of falling or to any special cause, but only to an indeterminate sense of fear, which overcomes the patient when he sees the place.

Other patients complain of an uncontrollable fear when they find themselves in a closed place with many men, as for instance in a theatre, in a concert hall, especially when they are seated in the middle of the auditorium, and they fear that they cannot get out, while this feeling is absent when they are seated near the door or in a box.

Others cannot travel on the railroad because when they are shut in the car they have the fear that they may not be able to get out. I have under treatment a gentleman who, not from fear of a railroad accident, but from fear of being shut in the car, for a long while has attended to his business, travelling many hundred miles in a carriage.

Others when on an elevation are attacked by great vertigo and the desire to jump off. This is only an excessive development of the vertigo from which many men suffer when on top of a tower or other height. To what degree this height-vertigo may attain, an otherwise healthy intellectual man has lately taught me, who for awhile lived on the second story, but recently has moved into a new large apartment two flights up and fitted with low windows. From his windows he looked out on one side on a square, and on the other on a broad street. From the first day he complained of a sensation of fear when he looked out of these windows, and this sensation soon became unbearable and lasted even during the night when he lay in bed. In the back room, the windows of which opened on a small courtyard, he did not have this sensation.

Many patients have sensations of fear when alone, and must always have a companion. This sensation leads at times to very unusual and even comical situations. A few years ago I treated a young neurasthenic physician, who could only attend to his practice when his old housekeeper attended him. When on one occasion he started on his visits without her, fear came over him when a quarter of an hour out of the city, and he was obliged to return.

Other patients live in constant fear of meeting their fellow-men—live as hermits in other words—walk along with sunken heads, and if they meet any one avert the gaze.

Others fear above all whatever they undertake or ask lest they lose or receive an undesired answer.

Others fear lightning or misfortune, or dust, or uncleanliness or some special material. These monomaniacal symptoms, which approach the boundary of the psychoses, we often see as the only phenomena in otherwise healthy individuals. I know a lady who spends almost the whole day in her dwelling sweeping away the dust, and washing her hands countless times. Another, the wife of the owner of an estate, is also affected by an overwhelming desire for cleanliness, is particularly shocked by wagon-grease, and personally inspects the clothing of her friends and relatives who visit her for traces of grease—a proceeding which has led to much bad feeling.

The fear of disease, especially of the contagious, is highly developed in many individuals. It approaches hypochondriasis. They watch their symptoms, read medical books and frequently resort to the physician to certify his diagnosis. Recent writers classify hypochondriasis with neurasthenia; indeed Arnot calls hypochondriasis a cardinal symptom of neurasthenia.

I will not discuss the question as to whether it is at all useful to give to each of these sensations a special name, as has been done by those who insist on systematic subdivision. Listen to these terms: agaraphobia or topophobia, fear of places; claustrophobia, fear in closed rooms; monophobia, fear of being alone; anthropophobia, fear of contact with men; mysophobia, fear of uncleanliness; photophobia, fear of light; pathophobia, fear of disease; siderophobia, fear of lightning; siderodrorophobia, fear of railroad travel; hypsophobia, fear of high places; batophobia, fear of falling from heights; pantophobia, fear of everything. You see, gentlemen, the number of terms may be increased indefinitely, and so such a scheme is of no utility. It suffices to recognize the chief varieties of sensations of fear from which these patients may suffer. They often constitute the most essential part of the clinical picture, throwing the other symptoms of neurasthenia into the background.

The symptoms which most frequently accompany the sensation of fear, are sensation of vertigo with pain and attacks of pain in the stomach, nausea, and fainting fits. These phenomena reach the highest grade when the patients cannot

determine the cause of their fear or seek to overcome it. Ordinarily there are present painful restlessness of the limbs, a purposeless moving here and there, a feeling of being alone and of exhaustion, of despair, transient heat and perspiration of the hands. Sleeplessness is added to the sensation of fear when this is lasting, and the patients cannot obtain rest at night. The fear increases in the dead of night in the imagination of the patient, his heart palpitates, he becomes hot, his skin itches, he is anxious, so that it is only towards morning that he secures sleep. These phenomena at times appear in perfectly healthy individuals as the result of great mental application or from care and trouble, but they disappear at once with the removal of the cause. In case of the neurasthenics, however, the sensation of fear and other phenomena are not related to the causes; indeed the cause is often entirely fictitious, only existing in the imagination of the patient.

It happens differently in case of the sleeplessness from which these neurasthenics suffer as the result of the years of work to which the brain has been subjected in association with bodily exertion and irregularity of life. Here the agrypnia is present in the sense of a deficit; sleep does not come, the patient is wakeful, without desire for sleep, until the morning hours, which bring a few hours of sleep. It is wonderful to what degree this sleeplessness may extend in case of male and female brain-workers without their ability for work suffering. Even when sleep only lasts two to three hours nightly the patient may remain for long fresh and capable. As for the length of time this may continue we cannot say, only the longer it is continued the deeper the influence. The difference in the action of hypnotic drugs in such patients is very interesting. While the usual hypnotics—morphine, chloral, paraldehyde, generally work very promptly in patients who cannot sleep from the presence of fear, the reaction of each individual to one or the other drug is different; some do not stand morphine and become chloral habitués, others only sleep under the influence of bromine. It must be noted, on the other hand, that patients who are only wakeful because they do not feel the need of sleep are ordinarily not affected by hypnotics to any noticeable degree. They lie quiet with a sense of comfort, but sleep does not set in, so that the attempt to cause these patients to sleep through medicinal agents is soon desisted from.

Of the spinal symptoms back-ache is the chief in the majority of patients. At times it is a pain located over one or another vertebra; again a vague pain in the whole vertebral column; again a painful sensation of weariness over the entire back, or of creeping, burning or pressure; again the pain accompanies motion; and finally it is worse when lying or sitting. At times spots painful on pressure are present, especially along the thoracic vertebræ or in the neighborhood; in other cases, however, pressure over the spinous processes evokes absolutely no pain.

The back-ache is ordinarily associated with a number of eccentric symptoms, and the name spinal irritation was formerly applied to it and a special disease made of it. To-day the symptom is included within spinal neurasthenia, and it is advisable to give up the term spinal irritation, since its use can only lead to error.

The clinical importance of the symptom back-ache is very slight to the physician, but to the patient, especially the hypochondriacal onanist and other neurasthenics, it causes great anxiety, since the amount and duration of the pain suggests the existence of an organic affection of the sacral spine. A careful objective examination will suffice to dispel this anxiety since all objective anomalies will be lacking. At times cases more difficult in diagnosis present themselves where one examination will not suffice to exclude an anatomical change in the vertebral column, especially tabes or myelitis. These are cases where, in addition to the rachialgia, there exist pain and weakness in the limbs, sensation of creeping in the feet, anæsthesia, impotence and lessening or increase of the patella reflex. Repeated examination will certify the diagnosis on the one hand from the inconstancy of the objective and subjective symptoms, on the other hand from the presence of the neurasthenic traits. A skilled neurologist will not fall into error in these cases.

The astonishing multiplicity of the sensible disturbances shows itself in general hyperæsthesia and hyperalgesia, especially a great sensitiveness of the skin to touch or pressure, to heat and cold, to changes in the weather, to many articles of clothing. We find further the manifold paræsthesiæ and anæsthesiæ in the skin and the deeper parts, sensations of heat or cold in the extremities, perspiration of the hands and feet

or abnormal dryness, creeping and itching of the skin, absence of sensation in the muscles.

Motor disturbances are either not present at all or only to a slight degree. There is a tendency to cramps in the muscles of the calf, sole, lower jaw in yawning, of the intercostal muscles on rapid turning in bed, of the abdominal muscles on sneezing or coughing. Paralyses are not met with, but the muscular system is rather very competent, and the patients who feel tired at home and worn out may walk for hours without fatigue.

Of the internal organs the heart, the stomach and the genital apparatus hold the front rank.

The nervous stimulation of the heart fluctuates markedly. The slightest excitement, and even this is not essential, causes tachycardia, and this is associated with a host of painful impressions, not alone the consciousness of palpitation, but also actual pain particularly in the neighborhood of the apex, followed by anxious arythmia with the most peculiar sensations before and after the systole, with a feeling of cessation of the heart's action, of beating, jumping, turning around, rising into the throat, etc. Often vertigo is associated with this tachycardia, or nausea, or sudden loss of strength, of death. Many patients of this character, when they consult the physician, have reached the diagnosis of organic heart trouble, and are much rejoiced when told that the physical examination reveals no anomaly of the heart. Taken all in all, heart-neurasthenia is the worst of all forms, owing to the anxiety and care it causes the patient. The very fear lest the heart should begin its irregular action causes the organ to work with double the normal beat. The automatic cardiac nerve centres are in a state of great excitability and the controlling action of the vagi is often greatly lessened.

The sudden effect of psychical causes on the frequency and rhythm of the heart's action is characteristic. An agreeable visit, pleasant news, etc., frequently brings the greatest palpitations to rest. I once treated a neurasthenic scholar who for months had suffered from arythmia of the heart, and a few hours after he had completed a journey to a pleasant winter resort he was free from all heart trouble and remained so for years.

The disturbances from the side of the stomach, including

to a greater or less degree those from the side of the intestines, are also manifold. They vary from loss of appetite and difficult and painful digestion, from simple distress during digestion to great pain during the process, nausea, great development of wind in the stomach and intestines, etc. Such patients consider the period of digestion the worst of their lives, while others, again, when the stomach is empty have cravings or pain in the stomach and intestine. Many are incapable of bodily or intellectual work when fasting, and regain strength only after eating. In others from time to time the appetite is voracious, and this leads to the ingestion of large amounts of food. A young man, whom I saw with Dr. v. Hösslin in his hospital, during the first part of his treatment did not touch his food till night-time, when his hunger was intense. An old, neurasthenic man of eighty years, who had not left his bed for years, at night when a voracious appetite seized him, ate in haste a dozen eggs and followed them with soup and other articles.

The appetite is exceedingly capricious; according to the subjective statements of the patients it may be lacking entirely, and yet they eat and digest a large quantity of food without special disturbance. The changeable nature of the appetite is a source of worry to the housekeeper and cook, particularly since idiosyncrasies in regard to certain articles and their method of preparation arise, which are again as capricious as the appetite itself.

The digestive troubles are in many patients quieted by muscular exertions, especially by long walks after meals. The well-known sense of well-being in the healthy and of rest after meals do not exist for the dyspeptic; his subjective troubles, etc., make him restless.

In this form of neurasthenia psychical effects are also of value. Joyful friends at dinner, and disputes after, lessen the disturbances, while a dinner alone, care, grief, etc., will increase them greatly. These over-anxious individuals eat scarcely at all digestible foods at home without after-effects, whereas at hotels they will run through the menu from beginning to end and digest the most indigestible substances, from which at home they would turn with disgust.

An effect on the constitution of these neurasthenic dyspeptics is in general exceptional. Only when the abstinence from

food due to the fear of digestive troubles increases, when habitual vomiting or aggravated constipation is present, does lack of nourishment show itself, and this at first sight makes the patient's diagnosis of cancer of the stomach plausible. Careful examination, however, yields a negative result, as well the physical exploration as the examination of the contents of the stomach before and after digestion, as also, eventually, the change in the symptoms and disturbances; and we conclude that only gastric neurasthenia is present and the result of treatment ordinarily quickly confirms the diagnosis.

The symptoms from the side of the intestines consist especially in an abnormal development of gas which, through the distention of the hyperalgesic intestinal walls leads to the most disagreeable sensations. This abnormal development of gas may be due to diseased innervation such as so frequently accompanies hysteria. The reason why the gas is not expelled, lies in the extreme distention of the intestinal walls in consequence of which the muscular coat loses its contractility. I have determined from experiments on dogs and on man that the intestinal wall when abnormally distended not only reacts on the motor impulse of the automatic centres, but also even on the electric irritability. Even when the road below is open the air in case of meteorism does not pass. Only after a part of the gas has been absorbed and the distention has been in part relieved do the automatic impulses act again, although after a weak fashion; at rather irregular intervals, about once every minute, a contraction wave starts from above downwards. The experiments on living man naturally only extended to the large intestines, which were blown up through the rectum, the anus being kept patent after the inflation process was ended by the insertion of a thick wooden tube; but since usually meteorism depends on a development of gas in the large intestine, these experiments certainly are valid ones. We are thus at least convinced why, when the conditions for gas development are present, the great distention of the colon is so lasting. Especially painful to the patients is the distention to the right and left behind; this is due to the collection of gas in the flexures of the colon, which of themselves constitute an obstacle to the downward passage of the gas.

As great as are the disturbances due to the gaseous distention, even so is the relief which follows the passage of the

gas. The intestine of a neurasthenic may work for hours to expel the gas; the aim accomplished there is a sensation of great relief, and then the head and spirits are light and the patient is again able to work.

The aggravated constipation which so frequently accompanies gastro-intestinal neurasthenia naturally increases greatly the meteorism, and the more so since the usual cathartics, even the mildest, are either not tolerated or work very unequally, and thus they cause new disturbances, and the patient soon abhors all laxatives.

In regard to genital anomalies which accompany neurasthenia, we must differentiate those which are causal factors and those which are the results of the neurasthenia. As a cause onanism and weakness of the sexual excitability in man and woman must be noted, with the difference that disturbances of intellectual working power affect still more the desire of man than that of woman. Male impotence, partial or total, is a frequent cause of neurasthenic weakness and of hypochondriasis. Aside from a few who, being unmarried, take little account of their impotence, being rather torpid in their sexual desires, man is very sensitive in this respect, and the moral influence shows itself to the expert eye in the entire individuality and conduct of the sufferer. It would carry me too far to enter on this subject further. The physician knows that much unhappiness exists in the world on this account. Perhaps in a later lecture I will give the result of my experience of and views on this subject.

Treatment.

The principles of treatment of neurasthenia are naturally evolved from what I have said in regard to etiology and symptomatology. We are usually dealing with a weakening of the innervation centres in individuals in good condition and often of strong constitution; not only the centres of will and thought, but also those of sensation, the automatic centres, the reflex and inhibitory. This functional weakness is combined with a high degree of irritability to external conditions in consequence of which the reaction on the above-mentioned centres and their functions is all the greater.

The treatment, then, must be principally a refreshing and

strengthening one to the nervous system, while at the same time it depresses the abnormal excitability. Irritants must be used and by means of them the irritability must be depressed, and this apparent paradox may be readily fulfilled if we use the milder irritants which do not increase irritability, and if we carefully accustom the system to stronger irritants by which we gradually replace the weaker. On the other hand, the injurious habits which the history suggests to us—the restlessness of the daily life, excessive work, care, excitement, family dissensions, troubled sleep, excesses, alcohol, these must be banished until the nervous system has been sufficiently strengthened to bear them without injury. Owing to the strong probability of the patients drifting back into their old habits, recurrence cannot be predicated against.

Removal from the usual surroundings and treatment in sanitariums, which are open for the special treatment of such patients and are conducted by competent physicians, is in many cases, especially the aggravated and chronic neurasthenics, necessary to obtain results. Milder cases, especially the pure cerebral form the result of overwork and in need of rest, may well be treated outside at some quiet resort where the patient may obtain good medical advice. I will state further on the essential external conditions. It is absolutely necessary to isolate and to treat in asylums the aggravated cases of the erethismal form of neurasthenia with disturbed constitution, loss of flesh, sleeplessness, dyspepsia, sensations of fear. Here no treatment is effective when the patient is at liberty, especially if surrounded by home influences. Many patients may well be treated at home, in the beginning, as every physician will note who is persuaded by his patient or the relatives to undertake the cure there, the novelty of treatment having at the outset a good effect, and the directions being followed. Soon, however, one or another direction is modified or neglected absolutely, the dietetic essentials are not attended to, and there is recurrence. Finally the treatment of the patient will only be partially carried out and in the end given up on the score that it does not benefit. In your own interests, therefore, gentlemen, as well as in those of your patients, you will be wise to undertake the treatment only in an asylum where there is every guarantee of medical observation and of the carrying out of even the minutest detail in treatment. In

Germany there are many good private asylums, and if the essential of the presence of a house-physician is lacking, a careful and intelligent nurse will suffice, and all intercourse with friends and relations should be forbidden. All expert neurologists agree that in order to secure cure it is essential to cut off all communication with the outside, and that the next most necessary factor is the securing of a skilled nurse who is a stranger to the patient. The well-known procedures of Weir Mitchell, which Charcot, Playfair, Biswanger, Burkart and others have recommended, and which have been of great service to me in difficult cases, are based on these precautionary measures. Absolute quiet of the brain and mind most quickly brings the disturbed nervous system rest, controls the abnormal irritability, as our daily experience with the psychoses teaches us.

Under such healthful surroundings the workings of the usual psychic and somatic agents may be thoroughly tested. Diet, massage, electricity, baths, drugs, can now be tested according to the individual requirements; especially may rest in bed be tested, a procedure which for aggravated myelasthenics is of the greatest value.

The majority of cases of cerebro-spinal asthenia do not require such absolute separation from the world, still for many of them partial seclusion in an asylum is proper. A certain amount of moral influence and a stringent medical supervision are not only desirable but also essential for patients whose wills are weak. This is proved by the fact that many patients who at first rebelled against the asylum later leave it unwillingly, and on recurrence of their complaint at once return. The certainty that the asylum and the careful medical supervision disperses their troubles, and the good effect of the physical agents there used, are remembered by them with gratitude after cure.

Many neurasthenics do not require asylum treatment; indeed such would not be advantageous to them. These are the individuals who are overworked; who are subjected to the monotony of daily work and the recurrent daily friction and do not possess marked resisting capability, and yet whose physical and mental constitutions are not deeply affected. Such neurasthenics, whose irritability is due to manifold bodily labors, are found in great numbers among the higher commer-

cial classes. These individuals recover quickly by a sojourn in the mountains in summer or on the southerly side of the Alps in the spring and winter, provided they have good nourishment and pleasant company. The unwonted exposure to pure air, the mountain climbing, the beautiful scenery, bring them relief, appetite and sleep, and in consequence they acquire quickly strength and weight and they feel sound and capable. They must, when possible, secure such change twice each year, in the spring after the hard winter's work, and in the late summer to secure strength for the winter. Thus a brain tested by severe work may continue to functionate normally.

The elevation of the mountain resort is not of the importance which is ordinarily claimed. Lofty resorts are all the year resorted to by thousands of neurasthenics in search of health, and in my experience there is no question that at Pontresina, St. Moritz, Samaden, etc., good results are obtained. We often, however, see the same results from sojourn in resorts of mean height, that is between two and four thousand feet, such as exist in the Bavarian Alps, in the Bernese Oberland, in the Salz Kammergut, in the Tyrol, and in Switzerland. In the choice of the proper place we must remember that it should not be shut in a narrow valley, but should be open, and that the service and the cooking are good.

In the winter and spring I advise resort to Meran rather than to the Italian sea-resorts, such as the Riviera. The strong mountain winds of Meran in March and April, of which consumptives complain, are far more suitable to neurasthenics than the enervating air of Italy.

I pass now to the consideration of the many neurasthenics whose circumstances do not allow of change of air and scene. With them the treatment must be instituted in connection with their home surroundings, and even here if both parties assist much may be accomplished.

Above all, gentlemen, I must impress upon you the necessity of individualizing. In no disease are the individual differences in the symptoms, both mental and corporeal, so varied as in neurasthenia. We must study each case separately in order to learn its peculiarities, and this consists above all in obtaining an exact history which will tell us all about the patient and his life. The interest which the physician displays by his careful inquiries has a good influence on the patient, and a

careful physical examination simply intensifies this. Since all depends on the recognition or exclusion of objective disturbances the examination must be most thorough. This requires much time both in the interest of certainty of diagnosis and in order to gain the confidence of the patient.

It is advisable after having examined the patient to tell him exactly the nature of the affection, the outlook as regards cure and the requisite treatment. All neurasthenics have the desire for information about their condition, and are very thankful to the physician for any light thrown on their symptoms.

According to the nature of the case you must now decide as to whether the patient may continue attending to his business during the course of the treatment or had best refrain for a greater or less time. The answer to this question is very important, since the entire result of treatment often depends on it. The physician must determine from the history and the examination the effect which the business and the family relations have on the patient. In case sleeplessness and inability to attend to business exist, then it is absolutely necessary to order the patient to abstain from his work. Each day that the worn-out brain is compelled to work only renders the situation worse. On the other hand, mild cases may well be treated while they are attending to their occupation by simply restricting the amount of work done. These hours of rest are useful for bodily exercise, varying with the sex, ability, and age of the patient. Walks, riding, baths, gymnastics, etc., are to be recommended. It is of great value to the young to persuade them to join a gymnasium; they soon become interested in the exercises, and association with their fellows drives away their hypochondriacal feelings.

Short journeys of a few days' duration to visit a friend, a large city, etc., are desirable; the new scenes have a beneficial influence. Travel of this nature I have found useful even in mid-winter.

It goes without saying that the bodily strength is sufficient to stand such exercise, and that the neurasthenic condition is not rendered worse by it. In aggravated cases we can only often determine this by experiment. Beard has pointed out with truth that myelasthenic patients in particular are rendered worse by tiresome journeys, by the lack of home

comforts and by the fatigue of seeking change of scene, etc. My experience accords fully with Beard's. Be very careful, gentlemen, how you order such journeys. Only the healthy should undertake protracted trips. For the sick, whenever it is desirable to change the scene, it is preferable to order resort to a place agreeable to the patient, but not far distant, where he may remain for some time and where he may undertake short trips according to his strength. If the patient feels well there, his abode and food being good, and the care given him being constant enough, then his physical strength improves, he gains in weight, he eats and sleeps better, while the disturbance of a journey to a distant place might simply alter his condition for the worse.

Many patients improve at the sea-side, and they obtain new strength from the sea-baths. Such patients are usually of strong constitution who are only suffering from a relative disability in their working-power. The shock of the bathing from the motion of the water and air, is borne as well as the monotony of their daily life and surroundings. In general it is not advisable to order sea-baths to the sleepless, excitable, or depressed neurasthenic. Their excitability and sleeplessness would be only thus increased. Such patients improve best amid the quietude of the mountains.

I desire to warn you, however, gentlemen, that it is often very difficult to decide as to whether the sea-side or the mountains is the proper place, and that we are often compelled first to test the effect of sea-bathing. At the outset it is best to recommend the still sea-bathing of the southern coast. If this answers, then the following year the strengthening baths of the north coast may be advised.

I pass now to the physical and therapeutic agents which may prove useful.

The physical therapeutic agents are of the greatest possible value in the treatment of neurasthenia; they may be used everywhere, in asylums or in private houses, although they are most effectual in asylums, seeing that there they may be resorted to after a methodical fashion and the best apparatus and personal skilled supervision is there obtainable. Still it is possible for patients who cannot give up their occupation to utilize certain of these agents at home or to go in the morning for a few hours to an institution where they may methodically

be resorted to. This will answer for the milder cases; for the more severe it will not.

Hydrotherapy is to be classed among the means by which the peripheral nervous system and also the nervous centres may be stimulated. All shock or too great heat must be stringently avoided; both are badly borne by an irritable, weakened nervous system.

The mildest but very effective procedure is rubbing with a moist sheet. This can be carried out anywhere and is not expensive; even the poorer class of patients may be subjected to it. It must be done every morning. At the outset, the sheet, which must be large enough to cover the entire body, should be dipped in water of the temperature of 24° R., and wrung out. The patient is to be wrapped in this sheet by the attendant, and any servant may act as such, and is to be rubbed from head to foot for a few minutes, and is then to be wrapped in a warm dry sheet and similarly rubbed.

I am aware that distinguished hydrotherapeutists are not in agreement in regard to the value of this rubbing, but I have satisfied myself hundreds of times that this mild procedure is essential at the outset to overcome opposition. Each succeeding day the temperature of the water is lowered .5° R. until 15° R is reached. To use colder water is not advisable in case of neurasthenics unless they happen to be individuals who are fond of very cold water. An addition of salt to the water increases the stimulant effect, but this should only be done at the end of two to three weeks. After the rubbing the patient should at once clothe himself and when possible spend a short time in the open air.

If the method has been rightly applied, then the patient should have a sensation of well-being, of warmth, of freshness and elasticity. In case the patient feels cold and uneasy, then at the subsequent rubbing a higher temperature should be attained. The method should be resorted to every morning for months. In many mild cases it suffices in conjunction with suitable diet to effect an entire cure.

Sitz-baths, cold douches preceded by packing, vapor baths of short duration, have an energetic effect. Such methods may be useful in case of the more torpid neurasthenics, but owing to the necessity of personal supervision they should only be resorted to in a retreat.

Full-baths of short duration, river and sea-bathing are very valuable adjuvants to treatment and cannot be too highly recommended. Many patients, however, cannot stand such forms of bathing, being chilled and light-headed after them. They should not be urged, but their treatment should be limited to rubbings, etc.

Electricity is very useful, as well the general application as urged by Beard and Rockwell, as the local in the localized phenomena of neurasthenia, especially those who suffer from weakened function of the heart and the digestive track.

General electrization after Beard's method was especially devised for neurasthenia, and the enthusiasm it created in America has been found warranted in Europe. The method consists in the application to the surface of the body of the positive pole while the feet rest on the negative pole, which consists of a metal plate wrapped in flannel. The positive pole has the shape of a roller, and thus it may quickly be passed over the skin. According to Beard and Rockwell the current should be strong enough to cause marked contractions of the superficial muscles. This advice I accept only *cum grano salis,* at least in the beginning of treatment. In my opinion the current should act only as a gentle peripheral nerve irritant; this is reflected centripetally on the central nervous system and leaves a feeling of general refreshment and strength, as also of sleep and appetite. If the intensity of the current is too strong at the outset then the unpleasant reactions set in which have been noted by the American authors: cold hands and feet, muscular pains and headache, general nervous excitability, etc.

These disturbances we can almost certainly prevent by beginning with a weak current, by not prolonging the séance more than five minutes, and by repeating only on alternate days. In case of very sensitive individuals it is a good plan for the surgeon to include himself in the circuit and to convey the current to the patient's skin by means of his own hand. This is certainly the mildest and best method of surface electrization.

The constant current used in this way has no special effects. It is much more useful in case of aggravated neurasthenia of special organs, such as the stomach, the intestine, the sexual organs, the heart, the head. Here the constant

current is of inestimable value as a sedative. In order to obtain the full effect, however, it is essential, and this has been apparent to me for long, to convey the current by means of large electrodes. The action on pain, on unpleasant sensations, on function, on the general health is, in the majority of cases, excellent, in many cases very striking. I cannot here enter into the details; in my article on "Electricity in Medicine" the reader will find details in regard to technique and action.

Massage is the third agent, which is as applicable as the previously mentioned agents and as useful at the outset, especially when combined with the others, massage and electricity alternating and hydrotherapy being used daily. In the beginning massage should be mild and of short duration (fifteen to twenty minutes) and gradually lengthened to one to two hours daily. We should begin with gentle kneading of the skin—that is to say, with mechanical irritation of the sensitive nerves. The intensity and extent of the rubbing and kneading should be gradually increased, so that the patient, after the procedure, has the sensation of being thoroughly aroused.

The action is very good and lasting. The sensitiveness of the body, the aches, the nervous disquiet, the sleeplessness will be influenced for good by massage, and the nutrition and assimilation of the muscles will be assisted. These actions are explained by the excitation of the sensitive nerves and the reflex action on the nerve centres, and also by the equalization of the blood and lymph streams in the skin and cellular tissue and muscles, all of which favor nutrition.

Massage is especially useful in the aggravated cases where rest in bed and freedom from work are absolutely essential. It plays a great *rôle* in Weir Mitchell's treatment. The voluntary muscles are absolutely re-enforced by massage, and retrograde metamorphosis is favored, without the central nervous system being in the least called upon.

In regard to the duration of massage and electricity, I advise their application from three to four weeks, and then a pause for one to two weeks, during which the patient may test his strength and acquire new patience for a continuation of the treatment.

The medicinal agents which are suitable for the treatment of neurasthenia belong to the class of sedative nervines, of tonics and of hypnotics. Do not, gentlemen, expect too much

from drugs in these cases. Only the bromides are apt to be very valuable in these cases, and then in large doses, 30 to 45 grains, repeated a number of times daily, in instances where nervous restlessness and sleeplessness exist. As the single remedy, however, the bromides will rarely suffice; they can only be looked upon as adjuvants. I order, when necessary, one or more teaspoonfuls of a mixture containing 750 grains of bromide of soda, and about six drachms each of bromide of potass, of bromide of ammonia and sometimes bromide of lithium, and I continue this prescription for a number of weeks if its action appears to be beneficial. Many patients like to order something for themselves in the midst of their complaints, and the bromides are useful in this respect. I tell patients who have sensations of fear or of impending ill, to carry in their pocket capsules of bromide, and to take them as soon as the sensation appears.

Patients who suffer from feelings of weakness, of hunger, and the like, I advise to carry with them and to take *pro re nata* a mixture of tincture of valerian, tincture of castoreum, and spirits of ether, about one drachm each. I aim thus less at benefiting the disease than at giving the patient the knowledge that when requisite he has a remedy at his command. To satisfy the cravings of hunger the patient may carry a little bread or similar article in his pocket.

As to the other nervines which Beard lauds highly—arsenic, cannabis indica, ergotin, caffein—I do not place much confidence in them. In exceptional cases they may certainly prove of value; usually, however, they will only answer the purpose of satisfying the craving of the patient for drugs.

Beard claims to have obtained results from the frequent change in drugs; one week he gives sedatives, the next tonics, the third he gives nothing, and then he begins over again. No German physician will lay stress on such practice, but we must remember that the North American likes and is accustomed to the taking of many drugs, while in Germany the custom which holds is the reverse.

The hypnotics, morphine, chloral, paraldehyde, I only use when absolutely necessary. I order patients who are sleepless to take once or twice during the week chloral or paraldehyde, but then I give them large doses so that a refreshing, quiet sleep may ensue. This plan often works very well. Then

the patients must for a day or so do without a sleep potion, and thus I continue until the general treatment induces sleep. Here I would note that often a warm bath or a mild galvanic current passed through the head at bed-time will assist in causing sleep. Others take so much alcohol at once that a species of drowsiness sets in. Beer in large amount tends particularly to cause sleep. An old neurasthenic gentleman who has been my patient for years has been able to sleep for a year only by drinking as soon as he gets in bed an entire litre of beer.

In case of gastro-intestinal neurasthenia, no matter how severe the pain—and indeed the complaints of the patients are never as severe as they claim—I must warn you, gentlemen, to beware of the use of narcotics, as also of digitalis and other heart remedies in cardiac-neurasthenia with arhythmia and tachycardia. If the attacks are severe, then a weak heart-galvanization a number of times daily will often work nicely, as also the bromides.

In general I also warn you against the use of purgatives, especially from the habitual use. Mild salines or other agents acting gently are occasionally, however, of benefit.

External agents, the so-called revulsives, are at times of value, although in general they are of bad effect. In case of intense and sharply localized neuralgias, especially rachialgia, they are allowable. Caustic ointments, blisters, the actual cautery do more harm than good.

Of great utility, on the other hand, are cold or warm applications, according to circumstances, along the spine, also the cold and warm douche over the sacrum, alternating every eight to ten seconds and protracted for about eight to ten minutes. The change in the temperature together with the mechanical irritation will often relieve the digestive disturbances of many dyspeptics and excite the appetite.

In conclusion, we must speak of the diet. Here it is difficult to lay down rules since the indication will vary with the case. Hence the method laid down by Weir Mitchell can only be followed in relatively few cases, especially since milk is not tolerated by many persons.

Nevertheless I cannot too highly recommend to you Weir Mitchell's valuable work, for from it you will learn how much food the stomach can tolerate and what results may thus be

obtained towards strengthening the system generally. Weir Mitchell's "Fat and Blood" and Beard's work on neurasthenia are indeed most interesting works. It does not follow, however, at all that I do not stringently warn you against so-called special system methods of treatment. If we could reckon the number of lives ruined by these cures we would be astonished at the high mortality rate. Cures of the kind in the hands of a physician who does not individualize or in the hands of the laity constitute a great danger.

ANTIPYRESIS

AND

Antipyretic Methods of Treatment

BY

DR. H. VON ZIEMSSEN

Director of the Medical Clinic, Munich.

ANTIPYRESIS

AND

ANTIPYRETIC METHODS OF TREATMENT

GENTLEMEN:

The advances in our knowledge of the causal factors of disease, for which we are indebted to bacteriological investigations, necessarily have altered our opinions in many respects and have opened up new routes in our therapeusis. Although for a long while the parasitic nature of the infectious material in case of the contagious diseases has been generally granted, still the determination of different species of bacteria and the results of recent studies in regard to their developmental processes have materially changed the whole aspect of the question. From the day when the specific germ was discovered dates without question the researches in regard to the biological behavior of the parasite in question, and in regard to the conditions which are capable of preventing its development and of checking or at least of modifying its action on the human organism. This opens up the prospect of an etiological or causal therapeusis, in the strict sense of the word, a prospect which for the moment overshadows all other questions in the medical sciences.

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eases, still clinical medicine must take account of this new factor, and in its light must undertake a revision of our methods of treatment of the infectious diseases. Many changes have thus imperceptibly occurred, and as our knowledge in regard to the carriers of infection becomes more precise, and their share in the genesis of disease phenomena becomes clearer, so in many ways will therapeutic applications become sharper and more accessible.

This applies at present in particular to surgical and to gynecological therapeusis, while internal medicine as yet is only busied with the practical worth of bacteriological investigations in regard to the etiology and the prophylaxis of diseases. In respect of these questions there is much dispute and useless theorizing, aiming at the overthrow of well-established fundamental facts in order to replace them by something new and hypothetical. As a proof of this agitation I need only refer to the doubts which have latterly arisen in regard to the usefulness of antipyresis and the value of the antipyretic treatment of febrile diseases. This question is of such practical importance that every clinician should take sides and report his observations in regard to it. Such has been the case already, as witness the transactions of the first and fourth congress of internal medicine.

The opposition to the antipyretic method of treatment arises from the old, recently resurrected view that fever is the curative reaction of the body against the causal factor of disease. This view of the salutary import of fever sprang from the very old representation of the cause of disease as a fight of the organism against a foe which had secured entrance, a fight in which the organism used all the methods at its disposal, especially those of fever, with greater or less effect. In the Middle Ages this view was prevalent over all others, and throve even into the middle of the present century. Virchow's opposition to this theory in 1850 is well known. His deductions favored in particular the opinion that the vis medicatrix naturæ was a power acting against the entering foe, protecting the body and seeking to eliminate the materia peccans. This view, deduced to a greater or less degree from pathological processes, claiming that there was a natural reaction of organic tissues against the harmful agent which had entered the body, was too broad a one, and therefore it has been ex-

ploded. We believe now that the effects of the action of harmful agents on an organ or group of organs or on the entire body are the necessary consequences of action on the cell and the consequent changes in the physiological function of the tissues.

With progress in research in regard to disease factors the influence of the regulating and compensating processes in the organism has sprung more into the foreground, and modern bacteriological research has also tended towards learning the importance and the effects of the regulatory processes. Although not so many years ago fever was considered an unwelcome—in the highest degree dangerous, under circumstances, indeed, a most deleterious—accompaniment of local or general disturbances, latterly a few observers have claimed, from a bacteriological stand-point, that fever was a useful—indeed, as regards killing of the noxious factors, a necessary reactionary element. From a theoretical point of view Samuel has busied himself with the question of the salutary importance of inflammation and of fever, those most important regulatory processes in the organism, and in particular with the influence which the bacteriology of to-day will exert on our knowledge of the causes and symptoms of disease.

From a clinical stand-point as well, our knowledge of fever, its influence on the organism, and the determination of an antipyretic method of treatment, have again become the cause of earnest investigation.

When the transactions of the first congress of internal medicine had given an impulse to the renewed investigation of the entire question, Naunyn above all others considered most carefully the importance of fever and the value of antipyretic methods, in particular hydrotherapy. This was in 1884, and the report of his investigations was followed by numerous publications from the pens of Winternitz, Murri, Maragliano, Gläser, Sassetzky, Symanowsky, Bauer and Künstle, H. v. Hösslin, Vogl, Fiedler, Port.

The different positions assumed by these writers on the question of antipyresis finally led to an important discussion during the proceedings of the fourth congress of internal medicine held at Wiesbaden in 1885. From the different publications it is clearly apparent that the opinion of clinicians and of physicians in regard to the importance of fever and in

particular of high temperature, and the necessity of energetically cooling the body in order to depress the overheating of the blood and organs, is no longer that held in 1860 by Brand, Jürgensen, Liebermeister and others, but that rather, indeed, in general, the extreme antipyretic view, which claimed that a lowering of the temperature was to be obtained at any price, has been relinquished; nevertheless, hydrotherapy for other reasons than those formerly held is still valued as the principal method of treatment, and antipyretic drugs are held in high esteem as adjuvants.

We may group authors in regard to their opinions of the worth of antipyresis, as follows:

The extreme hydriaticists, who only use the cold bath and reject internal antipyretics (Brand, Vogl, Winternitz, and others).

The moderate hydriaticists, who resort to luke-warm baths but reject internal antipyretics (Naunyn, and others).

Those who, according to indication, resort to moderate hydrotherapy and to internal antipyretics (Liebermeister, Jürgensen, Riess).

Those who consider fever a salutary and necessary regulator, and only resort to antipyretics when dangerous symptoms or complications ensue (Heubner, Curshmann, and others).

Those who claim that fever is a necessary phenomenon, and therefore reject all antipyresis and pay attention to diet only (Gläser).

Those who absolutely deny the influence of treatment on the mortality of typhoid fever (Port).

We see, then, that every possible opinion has its advocate, and that each advances, in his own opinion, cogent reasons for the view he holds.

It would appear, gentlemen, as if there were no agreement in regard to antipyresis, that there were no convincing facts, and that each may treat typhoid and other febrile diseases in accordance with his own views.

It would be a very regretable thing, gentlemen, if this state of affairs should result in great skepticism on the part of the physician, since this would necessarily lead to disastrous results in the treatment of our patients. It would be especially deplorable if, after a period of active antipyresis with its excellent results, we should reject this entirely on account of the

reaction in opinion, and return to the therapeutic indifference of the Vienna school. Against this all teachers, and in particular all clinicians, should strive, and it is a matter of congratulation that at the fourth congress of internal medicine the opinions of those who opposed absolutely antipyresis were at variance and carried but little weight. There can be no question but that the theoretical explanation of the necessity and usefulness of fever, offered from a bacteriological stand-point, is a plausible one and contains much that is true. The constant occurrence of fever in case of mykotic infection speaks in favor of the view that we are here dealing with a legitimate and to a degree with a valuable reaction. Nevertheless, we are familiar with a number of febrile infectious diseases in which suitable treatment not only abates the fever, but also the infection. I will mention only intermittent fever, acute articular rheumatism, the febrile attacks during the course of syphilis. Who, to-day, will expect in these instances a salutary effect from the fever? The experiments which were formerly made with the expectant-symptomatic treatment of intermittent fever, lend certainly no support to the view that fever is a useful phenomenon or that it has curative effects. We have here antimykotic specifics which play their appropriate *rôle*, and of these we have as yet only knowledge of a very few. In the great majority of infectious diseases, where specific agents are not at our disposal, we must assist ourselves by such means as experience teaches us can reduce the effects of infection, including also fever, to the greatest possible degree. The analysis of the action of the antimykotic or antifebrile method of treatment is certainly one of the most important questions of the day, but it is a question which cannot be settled in the laboratory of experimental pathology and of bacteriology, but rather at the bedside of the patient. The physician and the clinician must here make the distinction, and they should not allow themselves to be dislodged from their position by theoretical deductions and statistical data. No one treasures more than I do the additions to our knowledge of the causal factors of disease acquired through experimental, and in particular special bacteriological, investigations, as well as through reliable statistical data; still, observation at the bedside must be granted its full value in connection with the treatment and the cure of disease. Indeed such practical observation has

solved the majority of questions long before pathology has reached the determination of the connection between disease causal factor, symptom, and method of cure.

The question presented to us is: Has the antipyretic treatment of high febrile infectious diseases, and above all of typhoid fever, exerted favorable influence on the individual case? Has it had the same influence over the totality of instances, that is over the mortality rate?

We must consider each question separately. Observation at the bedside gives information in regard to the first, and the statistical data of clinicians in regard to the second question. We can state definitely that the course of the individual case under suitable antipyretic treatment has been more favorable and less dangerous, and again that the mortality rate of typhoid fever at least has fallen to one-half of the former rate.

Against this proposition various objections from prominent quarters have been made, the claim being that the lower mortality rate depends on factors other than the medical treatment. We speak only of typhoid fever, since in regard to it alone do we possess reliable statistics as to the course of the disease under different methods of treatment. These objections require special consideration, not only in order to prove them but also on account of the authoritative positions of the gentlemen who have advanced them.

On the one hand Fiedler claims that the lessened mortality of typhoid fever in the larger cities is due to the better sanitation, and he is unquestionably correct. Our own observations in Munich prove that the influence of good sewerage and the clearing of the ground and the cellars from effluviæ have been unquestionably of good effect. Nevertheless the relative decrease in the mortality depends more on the character of the epidemic than on the healthfulness of the dwellings, and markedly also on the nature of the treatment, as I will soon point out.

Port claims that the mortality rate is connected with the subsoil drainage. From statistics collected during the past ten years at the Munich garrison he finds that this drainage antagonizes rather the mortality than the morbidity rate, and that a decrease is an index rather of the severity than of the magnitude of the epidemic. He claims, hence, that the anti-

pyretic treatment is without effect on the mortality rate, seeing that the lowering of the earlier rate from 20 to 2 per cent., as is proved by the hospital statistics, would not be possible without the aid of a specific therapeusis.

Vogl, from a consideration of the same data, claims that Port's hypothesis is not tenable, so that we must await new data before accepting the latter's conclusions.

Vogl is the sharpest partisan of the exclusive hydro-antipyretic school which Brand founded. We cannot help granting recognition to the pure and energetic method of hydrotherapy in infectious diseases, since, surrounded by obstacles and objections, it has obtained position. Labors like those of Vogl are of the greatest value in the further development of the antipyretic treatment of typhoid, since the method was carried out in about the same class of patients, strong young men from twenty to twenty-two years of age under the care of disciplined attendants faithful in all details—that is to say, the bath was given at 14° R. of one quarter hour's duration, repeated every three hours, when the rectal temperature reached 39°, no matter what the season of the year, in unheated barracks with open windows night and day. Such constancy and care in his observations grants to Vogl's treatment the worth of an experimental study, and the results of these observations are marked enough. From 1875 to 1882, inclusive, the mortality rate of patients treated rigorously by Brand's method without the administration of drugs was 28 out of 520 typhoid cases; while in other divisions of the same military hospital, where a milder course of bath treatment was combined with the administration of internal antipyretics, the rate, during the same period, was 52 out of 610 cases. That is to say, the mortality under Brand's method was 5.4 per cent., while under a milder combination method it was 8.5 per cent. The conclusion is justifiable, then, that under the conditions within a military hospital Brand's energetic cold-water treatment proved superior to an antipyretic combination method, and that, all in all, it gave the best results as regards cure in typhoid fever.

With this conclusion the results obtained in the Prussian army, as published January 25, 1883, are in full agreement. From 1865 the mean mortality rate from typhoid sank from 25 to 8 per cent. of the affected, and this result is due chiefly to

Brand's cold-water method, although better sanitary surroundings and greater individualization in treatment assisted the treatment. The different mortality rate in the various regiments must be laid to the different treatment instituted, since it cannot be granted that a mild epidemic should only affect one regiment and a severe another. The large number of cases dealt with in this report excludes sources of error. The report states that the yearly morbidity in the Prussian army is 2000 to 3000. Of these 500 to 700 no longer die as previously, but only 160 to 240. It is claimed in the report that wider resort to the method of treatment would enable a larger number of the affected to recover.

These data are as free from local and individual influences as is possible. The Prussian army is spread over the greater part of Germany, in large and small cities, with good and bad barracks and military hospitals, the patients being treated by old and young physicians, the data extending over many years and the nature of the material for investigation being always the same.

There is lacking, however, a comparison of Brand's method with the modified hydrotherapeutic method and the combination antipyretic method. This is not the case with Vogl's statement of the results obtained from different methods in the Munich military hospital.

Although these statistics, when collected with the earlier publications of Brand, Jürgensen, Liebermeister, and others, prove that for military hospitals Brand's method is the most efficient, the same is not applicable to other people and places. Brand's method cannot in all its exactness be transferred to civil hospitals and to private practice. On the one hand there is lacking the trained attendants and the rigid discipline which renders impossible opposition on the part of the patients or neglect by the nurses; and again, and this I consider most important, the material in civil hospitals and in private practice is of a different kind, and not prepared for such energetic cooling as Vogl obtained as well through the temperature of the barracks as through that of the water. Recall, gentlemen, the circumstances with which we come in contact: Age, sex, developmental anomalies, pregnancy, anomalies in constitution, defective nutrition, unhealthy modes of life, drink, scrofula, tuberculosis, chlorosis, anæmia, heart diseases, syphilis,

etc.—such are traits and habits with which in the civil hospitals we have to deal in our typhoid patients in addition to the typhoid poison. The military surgeon should never forget this. We deal with different factors, and naturally enough, therefore, we must act and individualize differently in civil hospitals and in private practice from what is possible in military hospitals. These latter will have a lower mortality rate than the civil hospitals, since they contain as patients the young blood of the entire country, since all uncertain or bad constitutions are not admitted into the army, while we, in the civil hospitals, have to treat the dregs of the people. We consider, then, that our typhoid mortality rate of 9.6 per cent. in our Munich clinic out of a total of a little over 2000 typhoid cases in ten years, is very good.

Let us compare now the results obtained from the stringent cold-water method of Brand-Vogl with those granted by a pure and somewhat modified hydrotherapy in a civil hospital. The chief partisan of this method is Allen Naunyn. He does not, it is true, speak of a large material, but it is large enough to be cast in the scale. Naunyn lost by his modified water treatment 10 out of 145 cases, or 6.9 per cent., and this is certainly a good result. His method is the following: When the temperature in the axilla reaches 39.5°, and this is taken every three hours, the patient is bathed—that is, in general, he receives eight baths in twenty-four hours. As to the temperature of the bath, Naunyn distinguishes the cold (18 to 22° R., five to ten minutes' duration), the luke-warm (22 to 26° R., ten to fifteen minutes' duration), the warm (26 to 28° R.). The warm baths are given in the later stages of the disease, when the patient is delirious, restless, and where there is great muscular weakness. For years Naunyn has not used a bath the temperature of which was under 18° R. Internal antipyretics are not used. The diet consists of a litre of bouillon with an egg, or else 2 eggs with 1 litre of milk, 1 roll, 1 litre of water, or else 1.5 litre of wine or of grog consisting of .5 litre of rum, 50 grammes of sugar and hot water.

In addition to these groups of the extreme and moderate hydro-antipyreticists there is a small group of anti-antipyreticists who are opposed to all and any antipyresis. They treat typhoid according to the views of the Vienna school, that is by diet and expectancy. The chief partisan of this school

is Gläser, chief surgeon at the Hamburg general hospital. According to his statement he obtains at least as good results as, if not better than, the physicians who laud the methodical cold-water treatment. Gläser's records are especially valuable in that he has instituted a comparison of different methods of treatment of typhoid in the same hospital, with material of a similar nature and under the same attendants and dietetic rules. Careful examination of his records, however, reveals a fact which invalidates in part such a comparative estimate. He states that in his 102 cases water was used: 1. In the form of warm baths (27 to 28° R.) in case of sleeplessness and great restlessness; 2. In the form of the cold pack in case of aggravated bronchitis and pneumonia; 3. In the form of the water-bed. Gläser, therefore, has resorted to the cooling process, even though to a limited degree, since, aside from the heat-abstraction of the cold pack, we may look upon the warm baths of a temperature of 27 to 28° R. as not an indifferent procedure, since this temperature was always 5 to 6° under the actual blood heat of the patients. Such warm baths are often used under certain circumstances by some of the advocates of the cold water treatment—Naunyn, for instance—and they are unquestionably very effective. So long, therefore, as we do not know how often such baths were used, and as to whether the temperature of the baths was not occasionally lower (Gläser himself speaks of "hydropathic-impregnated assistants"), and further so long as we do not know how often the cold pack was used, and what method of treating typhoid was favored in the other divisions of the Hamburg hospital, so long we cannot grant great weight to these records of Gläser's. It would indeed be of value to possess from such a carefully managed hospital as the Hamburg a series of observations of cases treated after different methods, the results of which might be compared owing to the similar external surroundings of the cases. As yet, however, such has not been done.

Gläser's position approximates in a measure the method of Curschmann and of others. These writers treat mild and moderately severe cases of typhoid in general indifferently, and they only resort to means for checking the fever when the height and constancy of the fever becomes an element of danger, or when complications are threatening or present; and then warm or luke-warm baths are ordered rather than

cold. The adherents of such views are apparently to-day on the increase; whether with justice or not I will consider later.

We pass next to the group of writers who favor combined antipyresis, that is to say, the use of baths with internal antipyretics, according to indication in the individual case. This group seems to include the vast majority of clinicians and practitioners. Their published statistics agree completely, what we believe to be true, in the statement that typhoid in small cities (Kiel, Tubingen, etc.) in general has a lower mortality rate than that which prevails in large cities, either on account of the fact that the infectious matter is milder, or because the inhabitants of smaller cities and of country districts have a stronger constitution, and in consequence present a greater resistance to the infectious material than the inhabitants of a large city.

Within the limits of the combined antipyretic method there is room for modifications in the treatment, and it is not unlikely the method of these clinicians differs in detail in the hands of one and another. Cold, luke-warm and hot baths, the gradually cooled bath, the prolonged luke-warm bath, the cold sprinkling, the moderately cold pack, the cold pack, the simple washing, the internal administration of quinine, antipyrin, thallin, salicylic acid, kairin, hydrochinon, antifebrin, all these methods and agents have their friends and partisans, and each uses each differently from another.

You will now ask, gentlemen, what method of antipyresis may we in the future follow with our patients? What manner of hydrotherapy shall we resort to? What antipyretic agents shall we use, and under what circumstances shall we resort to them?

I will state briefly and tersely my position on this question.

Our answer must depend on a consideration of those changes which antipyresis should and can limit. As the name proves, the object of antipyretic treatment was at the outset deemed to be only the fever and the conjoined factors from which the fever originated—that is, the high temperature of the blood and of the tissues. This view, which probably originated with Liebermeister, arose from the supposition that the high temperature of itself, especially when lasting, was followed by a series of complications which endangered the life of the patient. The danger of overheating unquestionably is present

in case of hyperpyretic temperatures, etc., but it would seem as though high temperature alone had not the importance which Liebermeister granted it. Especially do the grave brain symptoms, the degeneration of the heart and of the kidneys, the trophic disturbances, seem to be not the effect of the high temperature, but coincident effects of the cause of the fever, and this cause is infection. We mean, however, by the term infection not alone the manner of origination of the disturbances by the infection-carrier, but also the diseased condition which arises from the presence of germs and their action on the organism. From the present stand-point of bacteriological investigation we must assume that it is not so much the microbes themselves which are deleterious as the products of decomposition which they originate in the animal tissues. The viability of the schistomycetes is limited in the human body, so it appears, not only by the taking of the material and oxygen necessary for their sustenance, but they also cause decomposition of the nutrient material through the development of unformed ferments and the formation of ptomaines or cadaveric alkaloids, which act as most intense poisons on the organism in which they are found. These destructive chemical processes which the germs cause both locally at the site of their first entrance, as also in the whole organism after they have increased and spread, we may for the time being consider the manner of the occurrence of infection.

If we ask now if against the presence of microbes and the chemical products which they cause, we are able to do anything of a positive nature, then we must deny this question as regards the majority of the infectious diseases. Specifics, that is to say agents which exert a special deleterious action on individual microbes, which, even when they have multiplied in number and extended throughout the whole body, may kill them or otherwise put an end to their life function, of these, indeed, we possess as yet but very few. The action of quinine on malaria, of mercury and the iodides on syphilis, of salicylic acid and of antipyrin on acute rheumatism, these agents we are correct in denominating specifics, but still we are in the dark as to the manner of their action, and even the so-called mykotic nature of the process, although it may be considered as proven for malaria, is for syphilis and acute rheumatism not as yet with certainty known.

Still another question imperiously pushes itself forward and requires the more stringently an answer, since it has blossomed for some time and has found adherents among physicians—I mean the question as to the salutary importance of fever. With the development of bacteriology the old teaching of the struggle of the body against the enemy which has entered arose again and the weapons of the *vis medicatrix naturæ*, the sharpest of which in olden time was considered to be fever, were again brought to the front. In this sense have distinguished teachers lately spoken "of the cleansing of the blood by fire," and of a "refining purgatory." Such explanations would be correct if either experimentally or clinically high temperature was found to have an effect on the life or capability of bacteria. This, however, as far as I can see, is not the case. It has been proved for certain species of microbes that a high or low temperature will kill them or check their development, but in case of the human body this temperature is either too high or too low. In case of the bacillus of typhoid Gaffky has proved that spore development proceeds at 40° C., although rather more slowly. According to Koch's experiments the tubercle bacillus ceases to develop only after exposure for a number of weeks to a temperature of 42° C., and the splenic fever bacillus loses its virulence but not its power of growth after a number of weeks' exposure to a temperature of 42.5° C., or to one of 43° C. for a number of days.

Experience has certified the fact that a temperature of 42° C., if long continued, is deleterious to the human body; we certainly cannot, therefore, claim that overheating of the blood is salutary in its effect.

A further factor, however, might still be active here, and this is the changes in the constitution of the tissues caused by the fever, which limit or take away the nourishing material of the germs. Clinical facts, however, do not point to the truth of this assumption, and particularly the fact that the acute infectious diseases in general run their course whether the fever be high or low, and, further, the progress of the infection does not stand in relation to the height of the fever. As a rule the severer fevers have the longest duration, and the slighter the shortest. If the fever were in any way deleterious to the bacteria then should it be considered advantageous. It has been claimed that in recurrent fever the extinguishment

of the vital functions of the spirochates was in connection with the extreme temperature, but still there are many other explanations for the death and disappearance of the spirillæ. I am rather driven to the conclusion that the life and action of germs in the human body, while in general these vary in duration for many species, are sharply limited, and that these are not dependent on fever, on treatment or on external circumstances. The duration of the infection period of typhoid is in general three weeks, of typhus fourteen days, of small-pox twelve to fourteen days, of scarlet fever six to seven days, of recurrent fever five to seven days, of measles five to six days, etc. It is well enough to know that there are many exceptions and deviations from the cyclical course, especially during great epidemics, but these deviations lie only in the direction of mildness and of shortening of the duration, without in the individual case the height of the fever being connected with the exceptional occurrence. In the abortive form of typhoid, of scarlet fever, of measles, of varioloid, the life of the germs is certainly of less duration and their action on the tissues of the body milder. The reason for this may well be sought in a weakening of their powers, and this may reside either in the biological properties of the germ or in special changes in the body in consequence of which the germ can only live a few days before it dies.

Of the greatest importance in the elucidation of this question is the sudden extinguishment of the vital functions of the microbes, which occurs at the time of the crisis. When in croupous pneumonia the fever crisis occurs 85 times out of 100 cases before the end of the eighth day, whether the fever has been high or not, still the pneumonococcus has the biological peculiarity of a longer life and action in the organism. Occasionally, of course, pneumonia lasts for from nine to thirteen days, but such anomalies also present themselves in case of other infectious diseases; further it is not as yet proved whether the microbe of pneumonia ambulans, intermittens, etc., is entirely identical with that of croupous pneumonia. In regard to the pneumonia which occurs as a complication of typhoid, of cerebro-spinal meningitis, etc., such an identity is highly improbable.

Opposed to these germs, which find their period of life within the cycle of duration of the disease, there exist micro-

organisms which remain for months and years active in the organism (those of malaria, of syphilis), and finally certain ones which, like the tubercle bacillus, remain for the entire life of the man in the body.

In case of those infectious diseases, finally, which do not cease at defervescence, we must claim that the entire generation of microbes does not end at one and a given time, but that certain germs remain alive for whose development the soil is favorable notwithstanding the lapse of the fever, as for instance in recurrent fever, in the recurrent typhoid, in malaria, etc. That in case of the majority of acute germs, sterility of the soil, complete cure, and immunity against recurrence sets in, is sufficiently known.

When we have solved these problems then will the answer to the chief question, What may antipyresis be expected to accomplish? be rendered easier. We must remember that we are dealing not alone with the checking of the fever and its effects, but also with the checking of the infection carriers and with their destructive products in and action on the organism. To draw the exact line between these two effects—that is, between the pure effects of fever and those of the microbes—is at present impossible. Naunyn's careful search through experimental and clinical literature has certainly cleared up many points, and altogether the majority of authorities agree in the truth of the preconceived postulate that the symptoms in acute infectious diseases are the minority due to the increased blood heat, and the majority to infection; but still much remains as yet obscure.

As the results of the overheating Naunyn only grants the acceleration of the heart and of the respiration. The disturbances from the side of the nervous system, of the blood and vessels, of the digestion, the tissue waste and altered secretions he claims are due to the infection. Most of the factors, therefore, are of a rather negative nature, and there remains much of a doubtful nature in regard to the *rôle* which fever plays in the nervous and circulatory disturbances. The instances of aseptic fever in which, according to Genzmer and Volkmann, aside from elevation of temperature, there is no further disturbance, assume, then, a rather exceptional rank. There are certain torpid natures, especially among the lower classes, whose nervous system is scarcely or not at all affected by

fever, who are able to work up to the last minute and only succumb when they are unable to make any further muscular exertion. We daily see such instances of walking fevers, especially the infectious fevers, both in adults and in children. Such cases are of special importance when the severity of the disease is later apparent with certainty. We made an autopsy a few years ago on a carpenter who fell from a badly constructed scaffold and was brought to the hospital in a dying condition. I found in addition to the fractures, the result of the fall, evidences of a typhoid at the end of the second week. According to the statement this man had accomplished day by day his difficult work as well as another.

In the same way many recurrences with high temperature are accompanied by no marked general disturbances. Children, in particular, in this connection, are very tolerant of fever. I have repeatedly seen in dispensary practice in children acute exanthemata fully developed and with no special fever, where the mother had suspected nothing before the examination. Instances of such toleration of fever are, however, exceptional.

In opposition to such instances are the cases where there is extreme sensitiveness to fever, where a temperature of 38° to 38.5° is associated with mild delirium and the sensation of severe illness. This extreme sensitiveness to fever is usually associated with the nervous constitution.

I mention these facts in order to show that the absence of symptoms from the side of the nervous system in case of certain febrile patients does not mean that the fever as such in general causes no nervous disturbances, but that they are dependent on infection. For my part I do not doubt that fever as such affects the nervous system, and to a greater or less degree according to the height of the temperature. On the other hand I deem it proved that brain disturbances may be caused by infection. In favor of this view speak those cases of deep affection of the brain functions, when the temperature is relatively low, for instance in typhoid, the sudden loss of consciousness and the delirium in typhus even in the first days, the great nervous symptoms in many cases of acute sepsis in the presence of scarcely any or of only slight fever.

Similarly the boundary between the effects of fever and infection on the circulation and respiration is a doubtful one.

That the quickening of the heart and the respiratory act depend on fever may well be claimed as proven, as also probably the relaxation of vessel walls, which is recognized by the constant appreciable dilatation of the arteries and capillaries, as shown by the pulse (marked dicrotism, disappearance of elastic elevation).

The relaxation of the vessel walls at the periphery, which is an accompaniment of fever, cannot be considered as due to a paralysis of the vaso-constrictor nerves, since the reaction on irritation of the skin by drawing the finger-nail over it, as Baümler taught, is very rapid. It is more likely due to an irritation by the over-heating of the vaso-dilator nerves, which are constituents of the warmth-regulating apparatus of the body.

If the above disturbances are caused by fever, then, on the other hand, are the severe nervous disturbances, the disturbances in the tissue change, the alterations of the blood and of the secretions, to be laid in great part to infection; as also heart weakness, the diminution in the number of red blood cells, the deficit of alkali and carbonic acid in the blood, the disturbances in the urinary secretion, the albuminuria, the trophic disturbances in the muscles and in the skin.

We will now consider the therapeutic methods and agents at our disposal against these disturbances, and seek in what way and to what degree they are able to exert a curative effect.

Hydrotherapy may well be considered at the outset of this investigation. In the opinion of all competent observers it holds the first place, since it combines all the requisites of a curative method, which in the matter under consideration are essential, and since in every respect it has an effect on the feverish organism. The good effects of the cold-water treatment have as yet only been noted in case of typhoid fever, since in other febrile diseases, such as erysipelas, pneumonia, recurrent fever, phthisis, etc., it is less conveniently applied. Nevertheless a good effect on the general state is to be expected in these affections also.

The action of the cold and the luke-warm baths may be expressed somewhat as follows: The bath cools the blood of the feverish patient, at first at the periphery and next by the circulation of the cooled blood from the skin to the interior of the body the internal organs are affected. Whether this cir-

culation of the cooled blood has immediate effects on the central nervous system is not proved, but I believe from a personal experience while suffering from a severe typhoid in 1884, when I was repeatedly bathed, that the desire for sleep setting in immediately after the bath, and which is its first effect, is to be laid to the cooling of the brain. This action, I must claim, is unspeakably refreshing.

Aside from this direct cooling effect comes the stimulation of the central nervous system, which I claim to be the most valuable action of cold baths. The stimulation of the sensitive nerves by the lower temperature of the bath is at once conducted centripetally, and this affects in greater or less degree all the essential nerve centres. The lowered vitality of the brain is stimulated, and from the refreshed centre extends a fresher stimulation of the nerves which preside over the circulation, the respiration, the digestive process, the locomotor apparatus, etc. The result is a quiet sleep, a clear sensorium, a refreshed look, easier movements, and desire for food.

We will consider separately the effect of hydrotherapy in these different directions. To begin with the betterment in the digestive functions, it must be stated that not only is there increased desire for food, but also that what is ingested is better digested than before. This speaks strongly for increased secretion of the gastric juice, and this can only be laid to a betterment in the innervation. We are able to nourish the patients differently, and this, in case of protracted fever, as in typhoid, is of the greatest value.

The circulatory apparatus is affected in a number of ways: Through reflex action, through the direct stimulation of the cutaneous blood-vessels, and finally through the stimulation of the heart muscle by the cooled blood circulating through it. The effects in their order are the following: The first obvious action of the bath is contraction of the peripheral vessels, often to such a degree that the pulse is imperceptible (this action being direct); next, increase of the blood pressure and over-filling of the vessels in the internal organs; after a while relaxation of the peripheral vessels and unchanged continuance of the high blood pressure. The heart works more slowly and more strongly, the walls of the vessels become more elastic, as is recognizable by the diminution of the dicrotism and the return of the elevation wave.

The peripheral irritation leads to deeper and more prolonged inspirations. The improved innervation leads to reflex cough, whereby the stagnant secretion is expelled. Thereby the tendency to bronchial obstruction, to atelectasis, to catarrhal pneumonia, is better checked than by the practice of turning the patient on his side from time to time, a procedure which I believe to be of purely theoretical value, in the first place because no typhoid patient will remain for long in the side position, and again because the early view of the mechanical production of hypostatic pneumonia is, I think, the wrong one. What is necessary is not the side position, but deeper inspirations with filling of the lower and dorsal portions of the lungs, as also the normal sensibility and reflex irritability of the bronchial mucous membrane, in order to prevent accumulations of mucus and the consequences of blocking of the bronchi—atelectasis and catarrhal pneumonia.

The secretions of the body are increased by the bath, that is the urine, the saliva, the perspiration, the tears. The tongue becomes moist, the eye clear, the surface soft.

The trophic sphere is also favorably influenced, as is evident by the non-occurrence of decubitus, etc. Aside from the trophic innervation the alterations in the circulation (blood pressure, lowering of the circulatory rate, stretching of the blood-vessels) play an important *rôle.*

The manner, duration, and temperature of the bath will vary according to the indications in the special case.

The full bath must be considered as the most effective, best applicable and pleasantest form of hydrotherapy.

The temperature of the water and the duration of the bath will be regulated by the stage of the disease, the elevation of the body heat, the quality of the heart's action, the condition of the nervous system and of the general constitution. In general we may say the earlier the stage of the disease the higher the temperature, the more resistant the heart and the nervous system; the stronger the constitution, the colder and the greater in duration should be the bath and the more frequently should it be repeated.

Very low temperature of the bath is to-day rejected by the majority of authors, and rarely is it used below 15° R.

I also must speak in favor of the mean temperature of the bath. In the majority of cases I order it, in accordance with

the constitution of the individual, between 18° and 24° R.; still, frequently in robust patients I order it as low as 14° to 15° R.

A bath at the temperature of the sick room is, it must be granted, very valuable. The patient should be bathed as often as is necessary during twenty-four hours in the same water, and this is renewed in the morning. In general a temperature below 15° R. is not essential.

The mean duration of the bath I consider to be from fifteen to twenty minutes. The number of baths to be administered varies from two to six in twenty-four hours, according to the intensity and resistance of the fever. I rarely am obliged to order a greater number of baths. The indication for a second bath is best guided by the temperature of the body. Brand's advice, to give the bath when the axillary temperature reaches 39.5°, has been considered bad, but in general I consider this advice good and I impress this on the attendants. Frequently enough cases are seen where the bath should be ordered where the temperature is only 39° R., and others where even a temperature of 40° R. does not necessitate it. Personally I watch the effect of the first bath in the reaction of the individual, the state of the nervous system, the pulse, and the subjective sensations. Of course, the effect on the temperature is also a valuable guide, but this should not be depended on alone. There are constitutions which are but slightly affected by cold baths, who again and again will feel refreshed after the bath, will have a good pulse and go to sleep, although the decrease in temperature is very slight and transitory. In others, especially those of nervous constitution, the action of cold baths is often bad. The temperature falls for a few hours 2° to 3° C., but the patient is restless, chilled, and does not feel refreshed.

You must not forget, gentlemen, that in your private practice you must take account of the subjective sensations of your patient and of the feelings of those surrounding him, and that therefore where possible without injury the mildest methods should always be resorted to. The miserable state in which the patient lies for hours after too cold a bath, and his depressed appearance in the eye of his relatives, will not add to the reputation of his physician. In fact such a state of affairs is a mistake, for we should aim at a refreshing, stimulating effect on the nervous system and not at depression or

shock. We prevent this the more certainly the more care we give to the irritation on the surface of the body, and the more slowly we allow the cooling action to affect the deeper parts. From this stand-point, and in particular with reference to the demands of private practice, I recommended two years ago resort to the gradually cooled bath, and I can recommend this method to you warmly as the result of its application to over a thousand cases of typhoid and other infectious diseases.

The patient is placed in a bath of the temperature of 26° to 24° R., and this is 8° to 10° R. lower than the temperature of the body, and is therefore in this respect to be called cold; the water is thrown over his body by the hand or a large sponge. Gradually during this procedure cold water is added until the temperature has been lowered to 20° or even to 18° R., when the patient shivers or complains that he cannot endure colder water. He is then removed from the bath and wrapped in a warm woolen garment, without being first dried. Thus wrapped up he may rest for one quarter of an hour comfortably, is then dried, clothed, and allowed to sleep.

By this method of bathing we have under full control the degree of reflex action we desire to obtain, and we may judge at the same time of the effect by the objective state and subjective sensations of the patient. The cooling of the blood by means of the gradually cooled bath is no less, notwithstanding the absence of shock, than from my observations it is clear occurs from very cold baths, because the process may be continued long enough. And although as regards the lowering of temperature the effect is less from this form of bath and from warm baths, still I hold it is better thus than to do too much.

The objections from certain physicians to the gradually cooled bath I can only explain on the assumption that it was improperly administered. I would claim that every physician who has watched the effect of the different methods of bathing on the objective and subjective symptoms in case of a large number of patients, and who has also had experience in his own case, will not be in doubt in regard to the excellent influence of the luke-warm and the gradually cooled baths over these symptoms.

In case of warm baths from 26° to 28° R., degrees which approximate quite closely the normal body temperature, but in

feverish patients are from 6° to 8° R. below the blood heat and therefore there is loss of heat, but the irritation is slight owing to the slight difference between the temperature of the bath and that of the skin; still, the feeling of well-being and the quieting of the nervous system and the heart are present, even the former appears here in the foreground and the lowering of temperature is very slight. The warm bath is therefore specially applicable to those cases which the older physicians called *febris nervosa versatilis,* cases in which marked weakness is a phenomenon. These cases, in which the infection usually has a malignant character—and we meet with them as frequently in measles, scarlet fever and small-pox as in typhoid and typhus—stand the shock of a cold bath badly, but the action of warmer baths is a good one over the subjective and the objective symptoms. The occurrence of sleep is especially desirable. No other means can so quiet the brain. When the nervous system has been more or less quieted, then we may resort to the gradually cooled bath, and even to baths of a temperature of 20° to 28° R. The system stands these better now, and the loss of heat and the stimulating and refreshing effects appear before the pure quieting action.

It would be especially objectionable and bad practice to order the same bath-temperature every time and for all stages. It is in this respect especially necessary to individualize in order to use this valuable physical agent properly. Not only do different constitutions and different degrees of infection need very different temperatures and forms of bath, but the temperature of the bath must also be frequently changed during the course of the same disease. In case of a weak, nervous female first coming under treatment in the second week of a severe typhoid, you must study the state of the nervous system, the temperature, the pulse, etc., and this may teach you that you ought to administer only warm baths from 26° to 28° R., possibly only a brief sponging or packing. The action on the pulse, temperature and nervous system may be readily determined, and the next day it may be possible to attain as low a temperature of the bath as 24° R., and later perhaps as low as 20° R. three to four times daily. In the third week, however, when the spontaneous morning remissions begin to show themselves, two to three baths at a temperature of 26° R. may suffice.

Thus, according to the requirements of the individual case, do the indications in regard to the temperature, frequency, and duration of the bath alter. The sum of the symptoms, the entire clinical picture and the way in which it is influenced by the first carefully administered baths, and not the effect on the body heat alone, should be your guide. You will, gentlemen, as the result of observation and careful thought, very quickly reach certainty in regard to the indications for the temperature, frequency and duration of the baths, as also in regard to the value of the other methods of using water.

In regard to cold sprinklings I would further note that when not too cold in a warm bath they may be resorted to, and often with good effect, in the severe asthenic forms of the different acute infectious diseases. This applies particularly to those infectious diseases which develop rapidly—that is to say, severe scarlet fever and small-pox—where all the therapeutic methods must be grouped within a few days. It is in these cases that James Currie, the originator of the method, obtained the most marked results from hydrotherapy, and the records of this industrious observer in his little work on the action of sprinklings with cold sea-water, to which he resorted in his severe cases of scarlet fever, should be more carefully studied.

The simple wet pack, when cold, acts only slightly as a depressor of the body heat and as a stimulant, since the temperature of the pack soon approximates that of the body, and the effects are largely superficial. The pack cannot be looked upon as an important antipyretic agent.

Ice-bags, which we often use, are powerful local refrigerants, but generally they do not affect the body to a degree worth mentioning. They answer less the purpose of antipyresis than that of antiphlogosis in limiting pneumonia, etc.

As to the utility of antipyretic drugs the opinions of authorities differ widely, and latterly the tendency has been to dispense with them more and more in case of the infectious diseases. This opposition, I think, however, is carried to an extreme. The administration of antipyretics in private practice has perhaps been too common, seeing that every febrile rise has been deemed an indication for the administration of quinine, etc. The value of an internal remedy, however, should not be depreciated on account of abuse.

The fact that year by year the number of antipyretic agents increases, as also their quality, as far as we can judge, speaks well for their use. The recently discovered agents are better than the earlier, not alone in that the antipyretic action is more certain and with less likelihood of untoward sequelæ, but also in that they are effective in much lower dose.

To speak from data derived from personal experience, I must say that I deem antipyretic agents as very valuable adjuvants to treatment, and that they are indispensable to the physician. As to the value of the different agents my experience has convinced me that antipyrin, thallin, and antifebrin are superior to the earlier agents, quinine, conchinin, salicylic acid, hydrochinon, resorcin, and others. Antifebrin is not alone superior to thallin and antipyrin in that it is safer and almost entirely unlikely to be followed by unpleasant sequelæ, but also on account of the small efficient dose and the low price.

In regard to the method of administration of these agents, their immediate and consecutive effects, I will speak in my lecture on the treatment of typhoid fever.

THE TONGUE

AS AN INDICATION OF DISEASE.

BY

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THE TONGUE AS AN INDICATION OF DISEASE.

INTRODUCTION.

THESE Lectures are published as delivered, with only a few alterations, chiefly verbal. I had intended to have begun them with a brief notice of what had been written on the subject of which they treat, but was compelled by the pressure of other matters to limit myself to the few necessary references which are to be found in the text. I now prefix such a sketch as I had designed. It does not pretend to exhaust the literature of the subject, but rather to give specimens of the views held at different times. The condition of the tongue has not escaped observation even from the earliest, though the allusions made to it are often fragmentary and incomplete. I have been disappointed to find how many and how great medical observers, whose minds must have been stored with pictures of the tongue, each with a practical motto attached, have given us none of their experience in this respect.

I have mentioned in the text (p. 656) some remarks of Hippocrates as to dryness of the tongue, and I now introduce further details which have been given by this great observer.

Hippocrates makes frequent mention of the tongue; in this particular, as in most matters of observation, striking is his superiority to the writers of many succeeding centuries. He often refers to the tongue in fever generally as dry or parched, sometimes without other qualification, as in the case of one Nicodemus, who had a febrile attack and a parched tongue after a debauch—an observation which can be warranted by modern instances. In several cases of fever not further defined, in a fatal case of fever, in fever after labor, and in fever

with jaundice, dryness of the tongue is mentioned. One Philiscus died with what is exactly described as intermittent hæmaturia; he had intermitting fever, intermitting black urine and lividity of the extremities; he had also a parched tongue. In one species of ardent fever the tongue is stated to be rough, dry and "saltish," in another rough, dry and very black. A young woman who had an attack of acute fever, of which she recovered, is represented as having a tongue which was "sooty" and dry. The blackness of the tongue is of interest taken together with what we know of the black tongue of typhus. In a case of quinsy the tongue is described as red and parched. Again, in connection with quinsy, the tongue is described as having characters which we cannot but recognize as those of glossitis, dry, swollen, livid, hard, inflexible and likely to produce suffocation—a danger to be averted by bleeding from the sublingual veins.

Aretæus speaks of dryness of the tongue in connection with pneumonia, ardent fever, and epilepsy, and of the mouth as parched in diabetes on abstinence from drink.

Paulus Ægineta, whose date has been variously assigned between the fourth and seventh centuries, refers to the tongue, sometimes on his own authority, sometimes on that of others. He mentions it as parched in the plague, and rough with ardent fever and phrenitis. For dryness and roughness of the tongue, with which he is evidently very familiar, he recommends the local application among other things of oil of roses and honey, and advises that the patient should lie on his side rather than on his back, and should keep his mouth shut, because "keeping it open allows the moisture to dry up." Paulus has here anticipated some later writers, though, unlike them, he has not inferred too much from his observation. He describes inflammation of the liver in a manner which clearly indicates abscess, and says that the tongue with this is at first red and afterwards black. Rhases, in the tenth century, describes the blackness of the tongue which occurs with plague, a concomitant of the disease which has since been often noticed. Dryness and blackness of the tongue, together with unquenchable thirst, were witnessed by Dr. Hodges in the Plague of London, as recorded in his "Loimologia."

Avicenna, in the eleventh century, speaks like Paulus of roughness and dryness of the tongue as calling for local treat-

ment, Indian salt, oil of roses, quince-seed, etc., and advises that those affected should not gape or sleep supine, for both these things dry the tongue. "A tongue covered with blackness is not to be permitted, but is to be scraped and dealt with as already described, for otherwise noxious vapors will arise from it and cause inflammation of the brain." If for "inflammation of the brain" we read delirium, we see here an instance of what is not uncommon—a sound observation connected with a theory which is not so. The concurrence of a black tongue with delirium is probably an experience as widely spread as typhus.

On the whole, the allusions to the tongue in disease are fewer and slighter than would be expected, considering the variety it presents; for we can scarcely suppose that the ancient physicians were so unlike their successors as not habitually to look at it as an act due to the patient, or that they could fail to gather from it much more than has found expression.

Coming to later ages, I have referred (p. 656) to the theoretical view of Willis, that the glutinous secretion about the mouth in certain conditions was due to inspissation of the nervous juice, and its discharge into the mouth by the salivary channels.

Sydenham seldom refers to the tongue, and never, so far as I can find, excepting simply to describe its aspect apart from theoretical considerations. He describes the tongue in the natural course of continued fever as neither dry nor very different from its usual color except in being rather whiter. If, however, the patient became heated beyond the usual amount of fever, the tongue then became dry and yellowish-brown.

Boerhaave, who wrote in the early part of the eighteenth century, and whose works amount to an encyclopædia of medicine and the allied sciences, makes occasional mention of the tongue, but less often than might have been expected. He dwells, like so many other writers, on the tongue in ardent fevers, describing it as "yellow, black, burnt and rough," a condition attended with unquenchable thirst. Much the same conditions, inextinguishable thirst, with a tongue which is dry, foul, white, yellow, brown or black, more especially if there be a scurf or crust on it, and on the mouth and nostrils, are

enumerated among the signs of malignity in acute diseases. Frenzy, he tells us elsewhere, may be foretold by blackness of the tongue. Boerhaave refers, as his topics suggest, and with more soundness of observation than I have found in any preceding writer, to the relation of the salivary secretion to disease and to dryness of the tongue. "The saliva," he says, "continually flows into the mouth of a person in health, and nothing is a surer sign to a physician of disease in a patient than his having a dry mouth." Elsewhere he tells us that an inflammatory obstruction is indicated, and phlebotomy called for, by suppression of the sweat and spittle. But the mouth he says is made dry also by exposure to air, as in sleeping with the mouth open, and when the nose is stopped up.

Mason Good, in his "Study of Medicine," presents, with much learning and amplitude, the state of medical knowledge in the year 1822. He describes the tongue of scarlatina as red with elongated papillæ, and in the more severe forms as covered with a black chappy crust. In typhus the tongue is first whitish, then dry, livid and black, while the teeth are covered with black, tenacious sordes. With yellow fever the tongue is at first white tinged with yellow, then black. Thrush is described as belonging to hectic: the aphthæ in dysenteric fever in particular foreshowing imminent death. With regard to the relation of thirst to dryness of the tongue, which have been so often associated by the older writers, Mason Good points out that there is sometimes but little thirst though the tongue may be dry and encrusted to the roots, while thirst may be vehement when the tongue and fauces have no dryness whatever. But he does not ignore the existence of thirst together with dryness of the tongue, for elsewhere he connects great thirst, a parched tongue, and an acrimonious condition of stomach.

Armstrong describes the tongue of typhus as besmeared with an adhesive secretion like melted glue, the organ on the evaporation of the thinner parts of the secretion becoming dry and varnished, later brown and then black, from the deposition, as he thinks, of carbonaceous matter.

Among later, but still bygone writers, the tongue finds mention more frequently than can be specified, with a general tendency to regard it, for diagnostic purposes, as little else than an exposed sample of the alimentary canal. There has

been a lack of endeavor to look at its manifestations otherwise than empirically, or to analyze its changes and educe laws from them.

Dr. Marshall Hall, in the year 1835, in the "Cyclopædia of Practical Medicine," treated of the tongue in relation to symptomatology. He gives without explanation the empirical knowledge or beliefs of his time, associating the white and loaded tongue with acute *synochus;* the dry brown or black tongue, the mouth being also dry, with typhus; a loaded tongue, of which the load peels off, leaving a red smooth surface, with acute dyspepsia; a furred dry tongue, with local irritation, or inflammation. He adds, with apparent originality, that a relation is to be discerned between the tongue and the secretions of the mouth and the state of the skin, a loaded tongue in particular going with a swarthy complexion and an oily condition of the skin, and a clean or smooth tongue with a natural state of the general surface.

Piorry, in the year 1837, endeavored to supersede the existing views by a system which largely consisted in the exaggeration of the drying action of the air upon the tongue. This, as has been seen, had by no means escaped the notice of his predecessors in ancient and modern times, though they have not carried their inferences beyond the bounds of discretion. Dryness of the tongue is due, in his view, to the abnormal passage of air over its upper surface connected with openness of the mouth, due to obstruction of the nostrils, enlargement of the tonsils, or many conditions of dyspnœa. He instanced localized dryness of the mouth as the result of the admission of air in the interval left by a tooth, and concluded that the coating of the tongue was chiefly the residuum left on the evaporation of the saliva. The old views empirically associating various phases of coating with derangements of the stomach and liver, blackness with adynamia, an ill-smelling coat with putridity of the humors, and so on, are to be replaced by a simple scheme in which the drying influence of air and the cleaning influence of food are principally concerned. To chew dry bread, says Piorry, is often a better way to clean the tongue than to swallow emetics and purgatives. The coat is simply dried saliva, which has been allowed to accumulate, and has no special significance. These views, in which truth and error are mingled, but of which the outcome is to ignore or attach

little importance to the tongue as a symptom, have apparently suggested much of the scepticism of modern times, in which both truth and error may be discovered.

Dr. Billing, in his well-known work on the "Principles of Medicine" (4th edit., 1841), reflects the belief of his time in supposing that the condition of the tongue reflects that of the alimentary canal. The tongue, says Dr. Billing, "affords evidence of the state of the mucous membrane of the stomach and bowels, with which it is continuous." He speaks of the tongue as naturally white before food, red after, and attributes the difference to the participation of the tongue in the condition of the stomach as regards its blood-supply, the tongue being pale from anæmia when the stomach is anæmic, red from congestion when the stomach is congested. In febrile diseases, such as early pleurisy, the tongue is pale from anorexia. The tongue pale from this cause does not call for purgatives. The tongue is red when the membrane of the *primæ viæ* is congested, as with dysentery, tuberculous ulceration, and typhoid. Dryness of the tongue, which Dr. Billing notes in connection with advanced febrile states, is due, in his view, to evaporation.

I must give a word to an excellent article on the tongue by the late Dr. Hyde Salter, in the "Cyclopædia of Anatomy and Physiology," 1847–1852. This is chiefly anatomical, but has an interest for the present purpose in the correct description which is given of the *fur* or *coat*, for Dr. Salter uses the term *fur* in this sense. "The *white fur*," says he, "consists of a white, opaque, soft, sodden epithelium, which, when viewed under the microscope, differs from the epithelium in its ordinary state in no other particular than its opacity."

Dr. C. J. B. Williams, in his well-known "Principles and Practice of Medicine" (3rd edit., 1856), describes the tongue as influenced by causes which act through the system, and so made red, furred, brown, dry, etc. More wise in his generation than some who have preceded him and some who have followed, he says that "the connection between febrile and other diseases and the appearances which they produce on the tongue, is not well understood: but the appearances probably depend on changes induced in the secretion of the mucous membrane and adjoining parts."

Copland ("Dictionary," vol iii. part ii., 1858) expresses the

opinion of his time in a laborious article, which contains the accumulated results of experience, in which he connects various states of the tongue with various disorders and conditions of system, but makes very little attempt to show why such associations exist, or by what means the several lingual changes are produced. He dwells upon the unfavorable import of dryness, and the contrary indication of a return of moisture. He acknowledges that breathing through the mouth may make the tongue dry, but only in the presence of febrile disease; and he refers to the diminution of saliva which disease of this kind engenders. He describes the *white, clammy,* and *loaded* tongues in a manner which conveys much truth but little instruction, since he associates them, with little endeavor to distinguish or explain, with most of the lesser disorders to which flesh is heir, including the less severe catarrhal, inflammatory, and febrile diseases, functional disorder of the digestive and respiratory organs, bilious derangements, visceral diseases, and smoking and drinking. With the *furred* tongue he is more explicit; he defines this much as I have done, describes the changes in it as due to elongation of the filiform papillæ, and the covering of them with mucus which dries, and connects the condition with severe inflammatory and febrile diseases, and with disease of the brain. He speaks of the exposure of a raw or smooth surface upon the removal of the *fur* as a bad sign, and of its becoming black and hard as a fatal one.

In the year 1863, Mr. Hilton, in his well-known lectures on Rest and Pain, gave evidence of, as he thought, a more immediate connection between nervous irritation and coating of the tongue than had hitherto been adduced. He pointed to unilateral coating of the tongue in connection with decayed teeth on the affected side, and attributed this to reflex nervous influence through the fifth nerve, and produced cases also in which the tongue had become unilaterally coated in connection with disease or injury involving the Gasserian ganglion or one of the branches of the fifth, in both of which circumstances he considered that the change in the tongue was due to nervous irritation. He adduced also the occurrence of grayness of the hair on the side of a decayed tooth, and on that of neuralgia, in evidence of local nervous action upon nutrition. It will be presently seen that by whatever means the grayness

may be produced in such circumstances, there are those who maintain, and apparently with reason, that the increased covering on the tongue is in most cases the result of the restriction of mastication and of friction on the tender side. I shall presently relate a case which suggests that injury to one chorda tympani may produce one-sided coating by interfering with the secretion of saliva on that side.

Dr. Martyn Paine, in his "Institutes of Medicine," published in New York (8th edit., 1867), generally connects the state of the tongue with that of the alimentary canal, and dwells in particular on redness of it as indicative of inflammation in this position. He considers that the changes displayed by this organ in abdominal disease depend on reflex nervous action.

Mr. Butlin, in the year 1879, attacked the belief then general that the "fur" of the tongue is chiefly epithelial, and endeavored to show that it consists mainly of vegetable organisms. Mr. Butlin uses the term "fur" in the general sense of coat or covering, and maintains that it is composed in health and in disease chiefly of minute living organisms (*schistomycetes*) and not of "cast-off epithelium." He supported this thesis by numerous examinations, mostly of scrapings from the surface of the tongue, and shows that while these display epithelial cells, the micrococcus forms their greater bulk. I shall show hereafter that examination of the tongue in section has given to me different results. Mr. Butlin has examined the organisms obtained, as I have said, with a care I believe not before given to them, and concludes that these are the chief constituents of the "fur" or coat, and not "cast-off epithelium," as before held. In this view the organisms accumulate, and the coat forms, chiefly from the absence of mastication; they flourish in moisture and upon liquid food; they cease to grow, and finally come off, with dryness. Mr. Butlin points out that the organisms he has described so minutely have no special relation to individual diseases, but grow in obedience to conditions which are common to many. Of whatever the fur or coat may consist, I think there is truth in much which Mr. Butlin has adduced, but that he has not presented the whole truth will appear from what will follow. It is true that the white coat does not mainly consist of *cast-off* epithelium; but nevertheless it is essentially epithelial, its bulk being composed of epithelium which is still in connection with the

papillæ and which has undergone the horny transformation. No doubt organisms are found upon this in varying quantities, but they do not form the major bulk of the white coat. On the other hand, it will be presently shown that such organisms are presented with great abundance in the dry, thick, usually brown, covering of the encrusted tongue, of which it would be no exaggeration to say that they form a very important part. (*See* woodcut, page 630.)

To Dr. Frederick Roberts we owe an excellent article on the tongue, which appears in Quain's Dictionary, 1882. Dr. Roberts describes the "fur"—which term he uses, like Mr. Butlin, in the general sense of "coat"—as consisting chiefly of epithelium, with the addition of the remains of food, bacilli and micrococci. He considers that the increased production of epithelium which causes the "fur," is due to hyperæmia dependent either on the general condition of the patient or upon direct or reflex irritation; on the other hand, there are serious conditions, one of which, as we are subsequently told, is long-continued pyrexia, in which the development of epithelium is prevented, so that the tongue becomes red or raw. Dr. Roberts attributes much direct influence to the nervous system in the furring of the tongue, and points to the thickly-coated tongue of apoplexy, to the unilateral furring of neuralgia, and to the furred tongue of migraine.

Finally, Dr. Roberts collects the empirical knowledge which associates certain diseases and conditions with certain states of tongue. In typhoid he tells us the papillæ are enlarged, and there is a whitish or yellowish "fur," or possibly the tongue is red, smooth, and glazed, and refers to the belief that if this is tremulous the bowels are deeply ulcerated. With acute rheumatism there is a thick creamy "fur"; much the same in attacks of gout, in which the tongue is apt to be also brownish. With acute dyspepsia, catarrh of the alimentary canal, and hepatic disorders, the tongue speedily becomes furred; with chronic dyspepsia it is thinly coated or clean and raw-looking; with habitual constipation it is large and furred; in acute gastritis it is red, with a tendency to dryness; in chronic dysentery, red, glazed and fissured.

Mr. Jonathan Hutchinson,[1] from whom no one can differ

[1] A course of lectures on diseases of the tongue, delivered at the College of Surgeons. *Medical Press and Circular*, July 1883, pages 1 and 17.

without apprehension, accepts Mr. Butlin's description of the "fur" as chiefly parasitic, and considers it to be chiefly the result of rest, or want of friction. "In order that *fur* should be present," says Mr. Jonathan Hutchinson, "two conditions are necessary. First, that the tongue, or at any rate the furred part of it, should be well clothed with long, well-branched, filiform papillæ; and, secondly, that these papillæ should have been left at rest and allowed to collect upon their tips the well-known white and somewhat slimy substance, which, in a more restricted use of the word, constitutes *fur*. Until recently it was generally taught and believed that this slime consisted of sodden epithelial scales. The accurate microscopic investigations of Mr. Butlin have taught us that this explanation is only in part true, and that the chief constituent is of fungus origin," the fungoid elements being present whenever the white "fur" is, whether in disease or health, and of no pathological significance. "The conditions," continues Mr. Hutchinson, "which favor the development of fur on the tongue are briefly those of rest. If we can fully realize this fact, it will help us to the explanation, in a very simple manner, of most of the morbid conditions of the tongue met with in acute diseases. When the tongue is quiet, the papillæ grow freely, their hairs accumulate epithelium, and in the epithelium colonies of micrococci flourish. Large allowance must be made for individual peculiarity, for it is only in those in whom the filiform papillæ are abundant and large, that any development of fur can possibly take place. It is well known that most persons have more or less coated tongues before breakfast, and that the taking of a meal rubs off the fur and cleans the tongue." Mr. Hutchinson then points out that the tongue cleans first where most exposed to friction, and explains the unilateral coating sometimes observed with tender teeth as the consequence, not of reflex irritation, as had been thought, but of the absence of free mastication. Mr. Hutchinson, in conclusion, offers the following suggestions:

"We must avoid assuming hastily that the condition" (of tongue) "present has any connection with the disorder for which the patient consults us. Many patients have habitually a profuse growth of filiform papillæ, and great tendency to the accumulation of fur. In others the papillæ are curiously absent, and the tongue may look bald or rough. In others

the furrows may be well marked, and the peculiar fern-leaf pattern present, and yet these several conditions may imply nothing whatever as regards the patient's health.

"In cases in which we have satisfied ourselves that the conditions shown are neither personal peculiarities nor yet the consequences of previous disease, we ought next to inquire carefully whether any local conditions are present in the mouth which will explain them, and by no means jump to the conclusion that they denote disorder of the stomach or liver. If the tongue is dry we inquire whether the nostrils are stopped, and if it is sore we must examine the teeth, and ascertain whether from sharp, broken points, from stopping with amalgam, or accumulation of tartar, any possible source of irritation exists.

"If we have failed to discover in the mouth any cause for disease on the surface of the tongue, we must still hesitate as to suspicion of visceral or blood disorder, and ask whether it be not possible that some irritant may have been introduced in the way of food. There are many fallacies in this direction.

"Lastly, if we feel able to confidently exclude all local causes, and obliged to believe that the state of the tongue is in direct connection with the state of the bodily health, we have still before us the difficult task of deciding as to what the nature of the bond of connection may be.

"The state in question may still possibly be in no way symptomatic of another disorder and not in any degree consequent on it, but rather part of the general disease.

"Above all, we must be on our guard against believing that the state of the tongue is a trustworthy criterion as to that of the mucous membrane of the stomach, and remember that for the most part a furred tongue implies that no food has been eaten, and little more, whilst glossitis and gastritis are conditions which are naturally independent and but seldom co-exist."

If I were to criticise these remarks from my own point of view, I should say that they were incomplete rather than inaccurate. I do not accept with Mr. Hutchinson the parasitic idea of the white coat, but there is much which presents itself to me as of truth and value in his clinical remarks. But the story is left half-told; the negative part is copiously and accurately stated, the tongue is justly disconnected from the

stomach and liver, but the connections which it has, or seems to me to have, with certain constitutional states are not recognized. There is no reference to over-growth of epithelium as distinguished from its undue retention, or to the important condition of dryness from salivary failure. Mr. Hutchinson, as a surgeon, regards the tongue with reference rather to local than constitutional influences. If I have examined it as a physician, with an eye to general states, I trust I have not ignored or minimized the local modes of action upon which Mr. Hutchinson has so impressively dwelt.

I think it will be apparent that some of the earlier opinions which I have collected, empirical though they be, those especially which associate dryness of the tongue with constitutional disturbance, and attach importance to the coating of it or to its denudation, generally without any attempt to explain either, are more consistent with the truth of nature than much of modern scepticism, of which the purport has been to explain and explain away.

PLATES.

DESCRIPTION OF PLATE I.

Class 1.—Healthy tongue. The epithelial covering is fairly abundant, but not excessively so; the papillæ protrude, but at no great length, and show a little horny epithelium upon their tips, on which are collections of micrococci, to be recognized as dark blue spots. The Malpighian layer, distinguished as a dark blue line abruptly separated from the corium, is of normal thickness. The corium is nucleated but not excessively.

The section, which is tinted with logwood, was taken from the tongue of a girl nineteen years old, who died in a state of collapse shortly after an extensive burn.

Class 2.—Stippled tongue. This during life presented the isolated white points of coating which mark the condition to which the term stippled or dotted is applied. The section shows elongation of the papillæ, with an excess upon each of the horny untinted epithelium which presents itself to the naked eye as the white coat. The intervals are not filled up or coated over, so that the coat is discontinuous.

The section is tinted with logwood. It was taken from the tongue of a child who died of lobar pneumonia.

Class 3.—Stippled + coated. Since this presents a mixture of the characters of Classes 2 and 4, it has not been judged to require a separate illustration.

Class 4.—Coated white, moist. The white coating during life, and to the naked eye. was continuous, so as to warrant the term coated instead of stippled or dotted. The section shows a great excess of horny epithelium on the papillæ, while the intervals are more or less filled, chiefly with epithelium of the deeper variety, so as to give the aspect of an uninterrupted covering. The corium is not over-nucleated or materially altered.

The section, which is tinted with carmine, was taken from a case of granular kidney.

Class 5.—The strawberry tongue is not shown separately, as its microscope characters are nearly those of Classes 4 and 6.

×30.

1.
Healthy

×30.

4.
Coated
White
Moist

×30.

2.
Stippled
Moist

LECTURE I.

MR. PRESIDENT AND GENTLEMEN,—What has been written with regard to the tongue is fragmentary and discordant. Among many conflicting opinions the anxious inquirer hesitates as to which he should be guided by. Should he begin with the earlier writers he may find that the further he carries his studies into modern times the further he leaves behind him any glimmering of light with which he started. If he enters first upon the most recent, as being desirous to come quickly to the cream of the matter, without troubling himself with what is below the surface, he may hastily conclude that the tongue is a blank upon which further research will be wasted. But there is knowledge which is not of books. Many a practical physician is able to read in the tongue rules of diagnosis, treatment, and prognosis, though he knows not how he acquired the power. It has come to him as part of the tradition of the elders, an accumulation of experience which there have been few attempts, and those not very successful, to analyze or reduce to law. The labor which has been bestowed upon the pulse in recent times is in remarkable contrast with the neglect which has been the lot of the tongue. And this has long been so. The ancient arms of our College display a hand feeling a pulse; they would give to view the duty of the physician less incompletely did they also present a tongue for inspection. The College of Heralds must determine whether it should be gules or argent, or, in the language of heraldry as of medicine, should bear a fur. "Various and full of meaning," says a great writer,[1] one whose words must ever be received with reverence within these walls, "are the conditions and appearances presented by the tongue. A patient would think you careless or ignorant of your craft if you did not at each visit look at his tongue as well as feel his pulse." But of late the tendency in this matter has been to agnosticism: it has become the habit with many, and those not the least influen-

[1] Sir Thomas Watson.

tial, to look upon the changes which this organ displays as of less and less importance, to regard them as accidental rather than essential, as the results of unimportant local conditions rather than as inseparably connected with constitutional disturbances. "The tongue," said a great surgeon in my hearing, "belongs to the mouth in the first place, and to the general system only in the second." I should have put the general system first; but I will not anticipate my conclusions.

I am about to examine the subject with a fresh appeal to nature, and, putting aside for the present both ancient faith and modern scepticism, shall be content simply to collect the evidence of the wards and the dead-house, and let the tongue speak for itself. This is simple in theory, but in practice complicated, as will become sufficiently apparent, and on this ground I ask not only the indulgence but the patience of those who honor me by attending. It is not my purpose to deal with ailments local to the organ, but only with those changes which have their origin outside it, or belong to the system at large. I look at the tongue as a physician, not as a surgeon, and regard it as symptomatic of disease rather than as the seat of it. My first endeavor has been to make a trustworthy and usable classification. Many terms have been hitherto applied vaguely and in confusion; tongues have been described as *furred, coated,* or *dirty* almost indiscriminately, while there has been too often a failure to attach due importance to such signs as dryness and nakedness. To secure a working classification I prepared hardened microscopic sections of a number of tongues of many kinds (the tongues amounted to 109), and made, with the help of the camera lucida, the outlines which are now before the College. I was thus enabled to associate the minute and intimate changes with the appearances presented during life to the naked eye. Taking together both naked-eye and microscopic appearances, I arranged the series into what, if I may borrow from the botanists, I may call *natural orders,* using as guides chiefly such characters as appeared on minute examination to be important, and which at the same time were distinguishable with the naked eye during life. It is sufficiently evident that no subdivision excepting one connected with characters evident to common sight could have any practical utility. Thus the classification, though made with the help of the microscope, can be applied

without it. It might have been more accurate in some respects, more satisfying to the votary of pure science, could it have been based only on minute anatomy; for there are some changes, especially one—increase of the deep epithelium—which are not easy to be recognized without the use of microscopic sections. I may say more of this hereafter; putting it aside for the present, I have made a classification which I think will work in practice, in which the minute and essential particulars are conveniently bound up with obvious external characters. The amount and distribution of the superficial or horny epithelium is important, since this essentially constitutes the white covering which is so noticeable during life. Other characteristics are: the elongation of the papillæ; the presence of incrustation, by which the proper surface of the organ is concealed; loss or attenuation of the epithelial layers, by which the surface becomes red, bare or raw; and the quality of dryness: this last is of great importance, and it may be added that it is recognizable with the naked eye only.

The classification may be thus sketched: First comes the condition of health; rather a variable standard—an average state, rather than an absolute one. Next come the stages of addition; then those of subtraction. In the stages of addition, the epithelium increases more and more, and finally acquires a superstructure largely composed of foreign material. This is the maximum of clothing; it is succeeded by a process of divesture, under which the tongue may become not only naked but flayed.

In detail, the first stage is where the papillæ are separately capped each with a minute white patch, which consists mainly of a horny epithelium; this tongue I call *stippled* or *dotted.* As the covering increases, the spots coalesce, cease to be discrete, and become confluent, or at least appear so to the naked eye. To this degree the term *coated* is applied, as indicating continuity. The increased growths on the papillæ form the larger proportion of the surface; the intervals are more or less filled up by the deeper variety of epithelium and adventitious matters. The coat attains its highest development in what may be conveniently designated as the *plastered tongue,* of which the covering is thick, uniform and conspicuous, and often looks as if laid on with a trowel. The term *furred* is restricted to another acquirement—elongation of the papillæ,

which remain separate from each other, at least at their extremities, so as to give a shaggy look, or one suggestive of coarse hair or fur. The last stage of increase, one which may succeed upon the furred tongue, or ensue without its intervention upon the coated or plastered, is where the papillæ are concealed by an incrustation, usually dark and dry, by which the surface is overlaid. From this, as the climax of addition, the scale descends through processes of subtraction. The accumulation comes off to expose either a normal surface or one which is imperfect: the former generally when the crust shelves off gradually, the latter when it breaks away abruptly. Irrespective of the formation and removal of crust, there are other modes of waste and defective growth by which the surface of the tongue is swept clean, and its coverings attenuated or even abolished in parts, so that these become absolutely skinless. We now have the several forms of the red, denuded, and raw tongue, and with these the scale finishes.

Before dealing with these classes individually, I will say a word about the plan I propose to follow. I shall first describe each variety of tongue, and then mention the clinical conditions which have been found with it. With this in view, I have made a habit of arranging cases which have come before me in a tabular form, according to the state of the tongue, annexing at the same time other details. As I must content myself with producing only abstracts of these tables I may say that the particulars systematically noted were: the disease and its duration; the general state as to strength, prostration, and consciousness; the temperature of the body; the arrangements as to food and drink; observations relating to the bowels and stomach, to the nervous system, to respiration with regard to the mouth and nose; the presence of morbid discharges by diarrhœa, diuresis, or suppuration; the amount of the saliva, and the moisture or dryness of the mouth. My performance in this matter has fallen short of my intention. I had hoped to have made a complete compendium of hospital practice; but many cases, chiefly the less important, have escaped notice, so that my tables include only 366 cases; these must be taken as a sample of hospital experience rather than as hospital experience in bulk. They do not fairly show the relative frequency of each kind of tongue, for the more trivial were more often omitted than those which were striking or

×30.

7. D. Furred. Dry.

Lizars, Edin & Lon.

DESCRIPTION OF PLATE II.

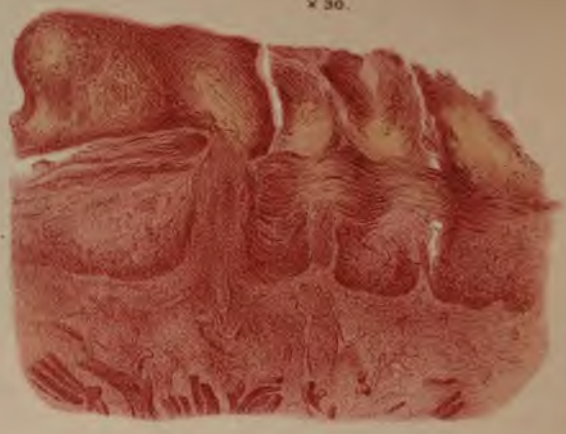
Class 6.—The plastered tongue. During life the tongue from which this section was taken presented the uniform abruptly-marked white covering like something laid on with a trowel. The section shows more elongation of the papillæ than in the preceding classes, while their summits are now somewhat covered, presenting an approach to the encrusted tongue to be shortly displayed. The papillæ are tipped with horny epithelium, much of which, together with extraneous and parasitic matter, overlies them and helps to form the thick white coat which is suggestive of plaster. The corium is not materially altered.

The section, which is colored with carmine, was taken on the eleventh day of pleuro-pneumonia.

Class 7D.—Furrred and dry. The tongue presented to the naked eye a long shaggy growth which covered its back part like long, very coarse hair. The characteristic of the section is the great elongation of the papillæ, chiefly by horny epithelium. The summits are not overlaid as in the encrusted tongue, which otherwise they resemble, but project as loose ends. There is considerable hypernucleation of the Malpighian layer and deeper epithelium and some of the corium.

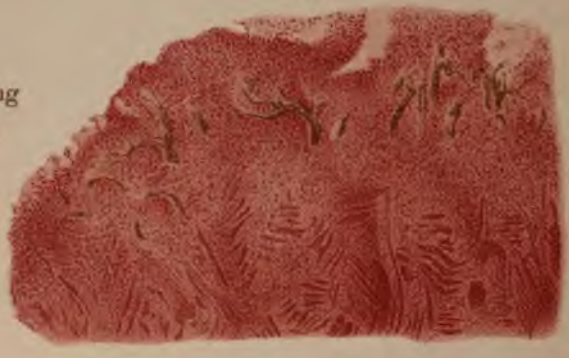
The preparation was taken from the tongue of a man, under the care of my colleague Mr. Haward, who died of sarcoma of the tonsil and larynx, and had long refused food. The section is colored with carmine.

× 30.



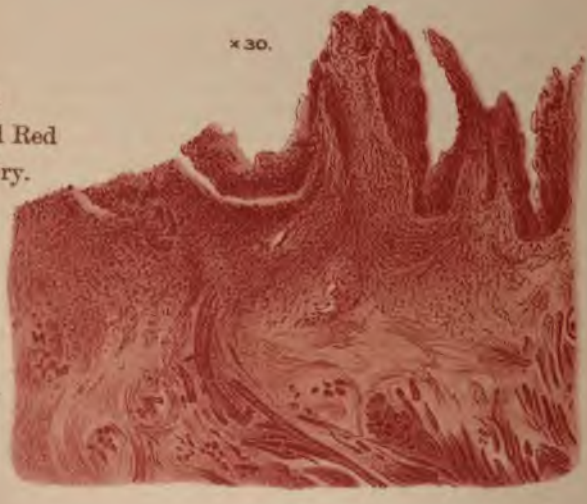
8.
Encrusted
Dry.

× 35



9.
Becoming
Bare.

× 30.



10.
Denuded Red
and Dry.

DESCRIPTION OF PLATE III

Class 8.—Encrusted, dry. To the naked eye covered with a dry brittle, generally brown, crust which conceals the papillæ. This variety is often presented in conformity with the preceding, as in some parts the elongated papillæ may have acquired or kept the accumulation, in other parts not.

The section shows great elongation of the papillæ, though these are obscured by the filling of their intervals largely with epithelium, while a confused mass, which higher powers declare to be partly parasitic (see woodcuts, pages 60 and 61), overlies all.

The section, which is coloured with carmine, was taken from a case of advanced phthisis with diarrhœa.

Class 9 shows one of the modes by which the tongue becomes denuded; rendered red and raw or partially so. This is by an inflammatory or congestive process connected with scarlatina. The corium is much injected and densely infiltrated with leucocytes. As a result of this process the epithelial covering has been extensively destroyed or dislodged; not entirely, however, for traces of it are to be seen in the portion displayed, while in other parts of the section more of the superficial investment was left.

The section, which is tinted with carmine, was taken from a tongue for which I have to thank Mr Sweeting, of the Western Fever Hospital. The subject was a child four years old, who died in the third week of scarlatina anginosa together with sloughing of the palate and nephritis.

Class 10.—Denuded, red and dry. This tongue to the naked eye was red, smooth, bare and cracked; much what is called the raw-beef tongue. The section shows in parts remains of the deep epithelial structure which elsewhere has been detached; a line of fracture shows at the junction of the bare and the partly covered parts between the Malpighian layer and the corium. The corium, which was widely exposed, is absolutely unprotected and in contact with the contents of the mouth. It is here densely infiltrated with small cells, probably leucocytes, and the remaining epithelium is over-nucleated.

The section, which is tinted with carmine, was taken from the body of a woman aged 44, who died of tubercular peritonitis with much thirst and fever.

CLASSIFICATION OF TONGUES.

TO THE NAKED EYE.	MICROSCOPICALLY.
1. **Healthy** **Moist**	White epithelium in small amount on papillæ, not continuous or superabundant
2. **Stippled, Moist** Dotted with white	Excess of white epithelium on papillæ, not extending between them
2. (D).[1] **Stippled, Dry**	Ditto
3. **Stippled+Coated; Moist** Coat continuous in parts	White epithelium on papillæ in excess, with partial filling of intervals
3. (D). **Stippled+Coated, Dry**	Ditto
4. **Coated White, Moist** Coat continuous	Excess of white epithelium on papillæ. Intervals more or less filled up with epithelium and accidental matters
4 (D). **Coated White, Dry** Coat continuous	Ditto
5. **Strawberry** Coated+injected, especially showing in fungiform papillæ	Like the coated or plastered, but with more injection
6. **White, Plastered** Thick uniform coat, abrupt and striking	More elongation of papillæ than with coated tongue, more filling of intervals with superficial accumulation
7. **Furred or Shaggy, Moist** Greatly elongated papillæ	Extravagantly long papillæ, mostly of horny epithelium
7 (D). **Furred or Shaggy, Dry**	Ditto
8. **Encrusted, Dry, Brown** Thick felted dry coat over papillæ	Continuous crust on and between papillæ largely of parasitic matters
9. **Furred or Encrusted becoming bare** Generally dry	Crust breaking away, together with more or less of normal surface
10. **Denuded, Red** Absence of normal covering	General absence of all epithelium excepting the Malpighian layer, sometimes of that also
11. **Red, Smooth, Dry, Membranous Covering**	Level membrane replacing epithelial processes
12. **Cyanosed**	Injected; hypernucleated; excess of deep epithelium

This classification is illustrated by the plates which are annexed; the figures attached to each drawing correspond with those in the table.

The letter D is used to imply dryness. Thus, to Class 2 a certain description is attached. Class 2 (D) presents the same characters with the addition of dryness.

considered interesting; but they show, I believe correctly as far as they go, the kinds of disease associated with each. However inadequate, I think these records will be of use, helped out as they must be with a larger amount of unrecorded observation. Having described each tongue with its environment of disease, I shall next regard the association from the other point of view, and, taking a few typical diseases and constitutional states, shall show with what varieties of tongues they are accompanied. Finally, I shall draw together such general conclusions and rules of practice as the foregoing details appear to warrant.

I will now proceed to describe the healthy surface of the tongue so far as is necessary for the purpose in view.

Class 1.—Healthy Tongue.

Plate I. No. 1.

The healthy condition of the tongue is by no means easy to limit or define. Not only does it change its aspect and character with often inconsiderable deviations from ordinary health, but it presents many differences within this state in different persons, and in the same person at different times. There are congenital varieties in the number and prominence of the papillæ, and there are such habitual differences, whether congenital or acquired, in the amount of epithelium, that to some persons it is normal to have a clean tongue, to others not less normal to have a coated one. Thus, whether the tongue be looked at with the naked eye or with the microscope, the range of health is wide; the same degree of coating may be normal in one person and abnormal in another, so that it is impossible to set up an exact ideal and say that all departures from it are the results of disease. Nevertheless, some broad outlines may be adventured outside which pathology begins.

The shape, color, and general appearance of the tongue in health are so well known that it is only necessary briefly to indicate what must be held to be consistent and what inconsistent with this condition. The shape is not too broad or the end too blunt, as with the flabby tongue of anæmia, of depression after alcoholic excess, and of many other asthenic conditions; nor is it indented with the teeth; nor is it narrowed and sharply pointed, as it is said to be chiefly with

acute febrile and inflammatory states. The tongue should be protruded steadily and kept still, neither jerky nor tremulous. In color it should present a basis or substratum of delicate pink, in which the forms of the papillæ are distinguishable by their shape, but not by hyperæmia or injection, which condition is conspicuous in many disorders, notably in scarlatina, as the chief characteristic of the strawberry tongue. The surface may be nearly clean or uncovered, but more commonly presents a superstratum which has been termed *fur* or *coating*, to the amount and character of which much importance has been attached. In ideal healthiness this is no more than a delicate white sprinkling or stippling, which may be likened to hoar-frost, a little speck lying as a cap or summit to each of the filiform papillæ, their extent not being enough to conceal but only to modify the general effect of the color below. The filiform papillæ are more constantly coated than the fungiform; the circumvallate seldom. But this covering, even in perfect health, is not always, even not often, so limited. Sometimes it stretches between the papillæ, partially fills up the intervals between them, and spreads more abundantly on the central and back parts than at the sides or tip. The coat is nowhere excessively thick, nor enough quite to conceal the configuration of the surface; it thins gradually without abrupt demarcation; it is not absolutely white, but is grayish where thin, yellowish where thick; the proper tissue, where exposed, is not of a bright but a dull pink, not exaggerated by injection, so that no striking contrasts of white and red are noticeable. The tongue and inside of the mouth are moist. Saliva can be voluntarily collected and spat out: it can be made to issue freely through a cannula in the parotid duct by placing a little acetic acid on the tongue.

To obtain an idea of the minute and essential changes which give rise to the altered appearances presented to the naked eye, it is not enough—it is, indeed, misleading—to be content with scraping the surface. It is needful to secure sections (see Plate I., No. 1) which show everything down to the muscular tissue; the amount, nature, and disposition of the epithelial investment must receive attention in its whole thickness, as well as the state of the corium with regard to injection, nucleation, and extravasation; and the characters of both apart from disease must be indicated. As the purpose is clinical, it will

be enough to describe the upper surface, where what may be called the symptomatic changes are mainly observed.

Starting from within, and using the terms descriptive of the skin, which the covering of the tongue much resembles we come first to the cutis vera, or corium, a layer of dense connective tissue between the muscular tissue and the epithelial. Projections from this form the central parts of the papillæ. The injection and nucleation of the corium are its chief points of morbid interest. The nucleation varies much, even in health; it is most abundant within the papillæ and near the surface. Upon the corium is the epidermis, of which there are three layers; the deepest is the rete Malpighii; upon this a stratum composed of lozenge-shaped nucleated cells, which correspond, with a difference, to the corneum of the skin; and upon this a horny formation which is special to the tongue, and chiefly forms the white coat.

To take these separately, the rete Malpighii presents in contact with the corium a compact arrangement of columnar cells, in health little more than a single course, which are distinguishable, though not abruptly, from a bulky collection of polygonal cells which complete the Malpighian layer. Towards the surface these cells elongate and flatten, still remaining nucleated, and with rather a quick transition become squamous and form a layer which varies much in different circumstances, by which the surface of the tongue is generally covered. This corresponds with the corneum of the skin, though in the tongue, unlike what happens in the skin, the cells retain their nuclei. On the prominent parts, where the growth is oldest, notably on the ends of the papillæ, the cells undergo a further change, losing their nuclei and cellular form and becoming fibrous, losing their power of staining with carmine, but greedily absorbing the aniline dyes. It is this horny epithelium which essentially constitutes the coat or fur, and to which many of the clinical characters of the tongue are due.

The superstructure has but a brittle connection with the rete Malpighii, a line of fracture often presenting itself with much regularity along their junction; on the other hand, the rete Malpighii is intimately attached to the corium, so that not only abnormal, but even exceptional, circumstances are needed for their separation. The Malpighian layer is a very definite and constant structure—the chief landmark anatomi-

×30.

11.
Red smooth
and covered
with Membrane

×30.

12.
Cyanosis

×30.

Excess of
Deep
Epithelium.

DESCRIPTION OF PLATE IV.

Class 11.—Red, smooth, covered with membrane. To the naked eye red, dry, and smooth. The section shows a common appearance of the red smooth tongue, the covering of the level surface with a membrane which helps to make it so.

The summits of horny epithelium have been swept away, and a new straight layer of epithelium of this class has been carried along the tops of the papillæ, partly supported by the recent epithelium with which the intervals are filled. There is some superabundant cell formation in the epithelium and corium.

The section, which is tinted with logwood, was obtained from the body of a young woman who had died of a discharging empyema associated with tubercular disease of the lung. There was, as in the preceding case, much fever of the hectic type.

Class 12.—Cyanosis. The chief characteristic of this section is excess of deep epithelium together with injected and hypernucleation of the corium. Some superficial or horny epithelium is seen towards the surface, but this is mostly covered by the excessive growth of deep epithelium. Collections of micrococci are to be seen on the surface.

The last section presents a condition to which a separate class has not been assigned, as it intermixes with, and is inseparable from, others. It displays, as does Class 12, a remarkable increase of the deep epithelium, not in this case connected with mechanical or obvious congestion, but with chronic albuminuria. A lesser degree of the same change in similar circumstances is seen in the section which belongs to Class 4. Some processes of horny epithelium are to be seen on the surface.

The section, which is tinted with carmine, was obtained from a case of old granular kidney with uræmia.

cally; what is above varies almost infinitely; the deep layer should not be quite bare, nor should the accumulation be such as to be level with, and so obscure the papillary eminences. The papillæ themselves commonly show each a tip of horny epithelium, white to the naked eye, yellow to the microscope. This varies much in health; so much in disease that the characters of coating and furring mainly depend upon it.

On the surface of the tongue, attached mainly to the prominences of the epidermis, is a varying amount of parasitic growth, chiefly the micrococcus. This presents itself for the most part in the shape of rounded accumulations upon the outstanding fibrillæ, like the inflorescence of a bulrush. Besides this, there are often seen, especially about the papillæ, granular heaps of bacteria and other parasitic matters, as well as detached epithelial cells and accidental matters derived from food; but the total bulk of the accumulation, whether parasitic or accidental, bears but a small proportion to the epithelial structure of which the coat or fur essentially consists. The adventitious growth is rather an appendage to the coat or fur than a necessary part of it. In certain conditions of disease, of which more hereafter, the parasitic growth, as well as the epithelial, may be in great excess.

The characteristics of health in the mucous membrane of the tongue may be thus summed up: The corium must not be over-injected or over-nucleated, nor must it present numerously the extravasation of leucocytes. The Malpighian layer must display no excess of proliferation. The middle layer of epithelium, which corresponds to the corneum of the skin, must be present but not superabundant. It should completely cover the rete Malpighii between the papillæ, but should allow these to be prominent at their points of emergence. The epithelial processes of the papillæ should be distinguishable, but not too long; upon these may be a few scattered points of vegetation. Finally, the surface described must be exposed, not concealed by any coat or accumulation, however derived, and it must be normally moist.

I need say little clinically about the healthy tongue. It will at once be seen that the healthy tongue is not the same thing as the tongue in health. There are individual peculiarities in the growth of lingual epithelium in virtue of which some tongues are always coated, others not so when coating might

be expected. With some persons a coated tongue is habitual, and not only consistent with health, but a sign of it. On the other hand, diseases like pneumonia, in which commonly the tongue is quickly and thickly coated, may fail to produce this result. It takes time to coat the tongue. In the table two cases of pneumonia are referred to with clean tongues. One was only in the second day and scarcely counts. In the other, a not very severe case of pleuro-pneumonia with a temperature of 102°, the tongue remained throughout so far clean that it could never be called otherwise than natural. It displayed a general fine white sprinkling, like April hoar-frost spread, which did not overcome the underlying tint. This absence of coat is quite exceptional in the circumstances, and probably shows habitual, but unusual, scantiness of the epithelial crop. There are also peculiarities in health which concern the saliva. An old woman, to be later referred to, displayed under capillary bronchitis a red dry tongue, from which I was disposed to augur ill. She got well and the tongue remained the same; she assured me that it had always been so, and I could only infer that the want of saliva, to which, as I shall presently show, the dryness and bareness of the tongue were due, was habitual and consistent with general health.

There are local and chronic diseases in abundance in which the tongue is normal; few involving pyrexia or any general disturbance. I shall show that this organ responds chiefly to constitutional variations. If the system at large is unaffected, so, as a rule, is the tongue. It is obvious that the conditions of observation in a hospital, whence my tables were chiefly derived, necessarily presented disease more abundantly than health, so that the morbid associations of the clean tongue are exaggerated.

1. Tongue not Abnormal.

Tetanus	1
Chorea	2
Sunstroke	1
Valvular disease	1
Intra-thoracic aneurism (dry diet)	1
Pneumonia (one on second day)	2
Pleuritic effusion (dry diet)	1
Pyo-pneumothorax	1
Phthisis	1

Whooping-cough	1
Ascites (hepatic): dry diet	1
Chronic albuminuria	2
Diabetes insipidus	1
Syphilis	1
Anæmia	1
Convalescent	6
(Total	24)

PARTICULARS RELATING TO THE ABOVE CASES.

Pyrexia (temp. from 102° to 104°)	2
Hyperpyrexia (temp. over 104°)	0
Temperature not recorded	1
Prostration	0
Chiefly on liquid diet	7
Diet strictly limited to liquids	0
Dry diet	3
Saliva abnormally deficient	2

Average temperature of 20 cases, 98.5°.

Died, 2; recovered, 8; relieved, 13; not relieved, 1

CLASS 2.—STIPPLED OR DOTTED TONGUE.

Plate I. No. 2.

This presents the first deviation from health. There is an excess of coat or fur, not uniformly spread, but displayed only on the points of the papillæ. The term *fur* is perhaps more suited to an interrupted or punctiform covering than *coat,* which may be limited to one which is continuous; the fur, however, is here but a mere beginning, which may develop into the coated tongue or that which will be described as furred or shaggy.

Before proceeding to minute particulars, the naked-eye characters may be briefly indicated. The tongue in this phase presents merely an exaggeration or accentuation of the covering of health; the state may indeed be so nearly that of health that the difference may pass without notice. The tips of the papillæ are covered with white more closely and broadly than within healthy limits, and there is perhaps a little more than the normal contrast between the white summits and the pink intervals. The term *stippled* or *dotted* fairly describes the aspect of the dorsum. To borrow from the nomenclature of small-pox, the acquisition is discrete, not confluent. The spots on the papillæ are usually white, less often yellowish or brown-

ish. The spotting is almost or quite absent on a narrow lateral margin, and also on a small triangular patch at the tip, the apex pointing inwards. These clear spaces are made so by the friction of the teeth at each side, and of the palate or gums at the tip.

The minute changes are to be made out only by examination in section. It is thus made evident that the essential alteration is in the amount and disposition of the epithelial covering. The change is in the way of excess, but the excess relates only to the condensed superficial structure, where the cells are flattened, fibrous, and rejective of carmine; not, or but very slightly, to the deeper parts, where the early shape and characters of recency remain. There is as yet no considerable or constant increase in the epithelium of the deeper sort, though there may be indications of a tendency in this direction; and here and there, but as yet not abundantly, excess of nucleation appears in the corium, especially about the papillæ. There is no marked injection of the blood-vessels. The intervals between the prominent papillæ are nearly empty, as in health. On their tips is commonly more or less parasitic growth, mostly the micrococcus, gathered, as it is apt to be, upon the projecting filaments. The proportion which these parasitic products bear to the total thickness of the coat or fur can be seen only in section. By mere scraping of the surface a false impression is given of the amount of these superficial products. The crop bears only a minor proportion to the bulk of the coat, the major part of which is epithelial. The parasites are the fringe of the garment rather than the garment itself.

We see in this tongue mainly the evidence of disuse. The length of the papillary processes may be explained by the absence of customary wear, and, indeed, is exactly what want of friction and scour might be expected to cause. There is little evidence of overgrowth in the shape of new epithelium, but only a retention of what must necessarily be old. In the parasitic formation there is nothing special or peculiar, but only what is found in health in less abundance. As with the epithelium, what is indicated is rather the retention of what is old than the development of what is new. We may attribute the accumulation, whether epithelial or parasitic, to the absence of food connected with loss of appetite, and perhaps in

some measure to the stillness of tongue which illness often engenders. I have referred to slight and early evidences of overgrowth of epithelium. This will become more apparent when I speak of more advanced changes.

This variety of tongue presents the first step in morbid acquirement and the first departure from health. A large number of persons could be found with this tongue whose condition is not abnormal. I have tabulated sixty-one cases in which this state of tongue was seen. All were hospital patients, in whom absolute health was scarcely to be looked for; among them acute disease was infrequent, chronic disease common. Heart-disease was more numerously presented than any other disorder, occurring in seven. Pneumonia and pleurisy, taken together, were found also in seven: but among these was only one instance of pneumonia in an active and pyrexial state. Rheumatism described as acute was present in five cases, but in only three was this adjective applicable in its fullest sense.

The general absence of pyrexia was striking: the temperature in only three cases reached 102°; in no case was it known to pass 103°. The diet in the large majority of cases comprised solid food. Three persons were kept for special reasons as short of liquid as was practicable. In these the saliva was markedly deficient—a deficiency which was noted in but four other instances. Extreme prostration existed in but one, together with acute obstruction of the bowel. Thus this tongue does not belong to pyrexia, and seldom concurs with grave constitutional disturbance of any kind. With the acute obstruction the temperature was normal. It presents itself often with local or organic disease, where there is little fever and seldom much vital failure.

Subjoining the cases where the stippled or dotted tongue was dry, and putting aside one where the dryness was due to diet, a larger proportion of acute disease and of constitutional disturbance is apparent when the tongue is dry than when it is moist.

2. Stippled or Dotted with White; Moist.

General paralysis or paresis	2
Chorea	1
Hysteria	2
Neuralgia	1

Valvular disease of heart	7
Pericarditis	1
Pneumonia, pleuro-pneumonia, broncho-pneumonia	6
Pleurisy	1
Laryngitis	1
Bronchitis	2
Phthisis	1
Tonsillitis (follicular)	1
Acute obstruction of bowel	1
Fæcal accumulation in colon	1
Epistaxis, nostrils plugged	1
Jaundice	2
Ulcer of stomach	1
Dysentery	1
Chronic albuminuria	5
Diabetes mellitus	2
Hæmoglobinuria	1
Acute rheumatism	5
Subacute rheumatism	2
Acute gout	1
Chronic gout	3
Anæmia	4
Lymphadenoma	1
Acute alcoholism	2
Dry diet (1 for ascites, 1 for pleural effusion, 1 for cardiac dropsy)	3
(Total . . . . 62)	

PARTICULARS RELATING TO THE ABOVE CASES.

Pyrexia[1] (temp. 102° to 104°)	3
Hyperpyrexia (temp. above 104°)	0
Much prostration (acute obstruction of bowel)	1
Prostration not severe (pericarditis)	1
Chiefly on liquid diet	10
Strictly limited to liquid diet (acute obstruction of bowel)	1
Dry diet (liquids reduced as far as possible)	3
Saliva abnormally deficient (including 3 under dry diet)	7

Average temperature of 42 cases, 98.5°.

Died, 15; recovered, 18; relieved, 25; not relieved, 3.

2 (D). STIPPLED OR DOTTED; DRY.

Pleurisy with effusion (dry diet)	1
Peritonitis (opium)	1
Acute obstruction of bowel (volvulus)	1
Chronic albuminuria	1
(Total . . . . 4)	

[1] The temperature was not noted in 13 cases; it may be taken in these as not materially elevated.

PARTICULARS RELATING TO THE ABOVE CASES.

Pyrexia (temp. 102° to 104°)	0
Hyperpyrexia (temp. over 104°)	0
No observations as to temperature	1
Average temperature of 3 cases	98.6°
Much prostration	2
Prostration not severe	1
Chiefly on liquid diet	0
Strictly limited to liquid diet (1 fed by bowel)	2
Dry diet	1
Saliva abnormally deficient	0

Died, 1; recovered, 1; relieved, 2.

CLASS 3.—STIPPLED + COATED.

The next step in the acquirement of coat is intermediate between the stippled and the coated, presenting a mixture of the characters of both. The white accumulation is present on the papillæ, but is not limited to them; at the back and along the median fissure it fills up the depressions, so that the tongue, while displaying the dotted character about the anterior and lateral parts, is at the back continuously covered, the continuity being implied by the term *coated.* This tongue is one of very frequent occurrence, perhaps more so than any other which can be regarded as morbid; it represents but a slight departure from health; since it partakes much of the class preceding, and of that following, it may be dismissed with brief description. A partial stippling may present itself as a stage either in the formation of coat or its removal; an example of the latter is seen in the drawing taken during recovery from pneumonia.

The microscope displays in a more marked manner the characters which have been described as belonging to the stippled class; the papillary processes are elongated and conspicuous, but there is in addition some increase of the deep epithelium by which the intervals are partly filled, the filling up made more complete by detached epithelium and accidental matters. Though the deep epithelium does not present to the naked eye the whiteness of the superficial or horny kind, yet the increase of the latter and the mixing together of the sloping ends of the papillæ give a continuity of whiteness, though only in the central and back parts, where the epithe-

lium is most plentiful. This tongue shows want of wear, together with more evidence of increased growth than in the preceding class.

Looking at this class clinically, we still find a large proportion of chronic disease, but note an increase of acute and constitutional affections. I may observe as an apparent exception that pneumonia is presented less numerously, though acute rheumatism is more so, and typhoid is now introduced. The change in the character of the cases is shown by the increase of pyrexia, though this is still slight compared with what will be seen presently, by the increase of constitutional depression, and by the number of cases in which the state of the patient was such as to necessitate liquid diet.

3. PARTLY STIPPLED; PARTLY COATED; NORMALLY MOIST.

Bulbar paralysis	1
Cerebral hæmorrhage	1
Cerebral embolism	1
Hemiplegia (cause uncertain)	1
Giddiness	1
Chorea	1
Hysteria	2
Valvular disease of heart	5
Pericarditis (not rheumatic)	1
Pneumonia	2
Laryngitis	1
Bronchitis	1
Asthma	1
General tuberculosis	1
Phthisis with hæmoptysis	2
Ulcer of stomach with hæmatemesis	1
Cirrhosis of liver	1
Ascites (cause uncertain)	1
Anasarca (cause uncertain)	1
Diarrhœa	1
Constipation	2
Colic	1
Chronic albuminuria	6
Acute rheumatism	7
Subacute rheumatism	2
Chronic rheumatism, or sciatica	2
Gout	3
Congenital syphilis	1
Anæmia, chlorosis, etc	4
Addison's disease	1
Ague	1

Typhoid	3
Erythema nodosum	1
Eczema	2
Pemphigus	1
Acute alcoholism	1
Abscess, internal, no discharge	1
Stricture of œsophagus	1
Convalescent	2
(Total . . . . 69)	

PARTICULARS RELATING TO THE ABOVE CASES.

Pyrexia (temp. 102° to 104°)	6
Hyperpyrexia (over 104°)	0
No observation as to temperature	14
Much prostration	5
Prostration not severe	6
Chiefly on liquid diet	23
Strictly limited to liquid diet (stricture of œsophagus, 1; diarrhœa, 1; constipation, 2; colic, 1; typhoid, 3)	8
Dry diet, liquids reduced as far as possible (ascites)	1
Saliva abnormally deficient (decidedly), including 1 under dry diet	5

Average temperature of 54 cases, 98.7°.

Died, 7; recovered, 30; improved, 26; not improved, 5; uncertain, 1.

CLASS 4.—THE COATED TONGUE.

Plate I. No. 4.

The next degree of epithelial excess, whether by retention or over-production, results in the formation of a continuous coat, or one continuous over the greater part of the dorsum. This covering presents great variety and many indications to the naked eye. There is scarcely an acute or sub-acute disorder at some period of which the tongue is not coated: this holds good especially with febrile complaints. This state of the tongue is often a step to other changes in it, those often of more serious import. Two special varieties of coated tongue will receive separate notice: the *strawberry* tongue, which, besides being coated, is much injected; and the *plastered* tongue, in which the coat is superabundant and has certain characters of recency. Putting these kinds aside for the present, the generally coated tongue is only an advance upon that which is partially so, just as the partially coated tongue is an advance upon that which is simply dotted.

In the ordinary varieties of coated tongue, the coat which is continuous between the papillæ covers the greater part of the dorsum, being thickest where friction is least, about the back and median furrow. It may be dirty-white, grayish, or yellowish. The limitation at the tip and edges is not abrupt; there are no striking contrasts of color; neither is the coat very white, nor where it is absent is the mucous membrane vividly red. The fungiform papillæ may be visible where the coat is shelving, but they are not remarkably injected as with the strawberry tongue. Such a tongue may be anæmic or sodden-looking, especially when the patient is anæmic, the condition chronic, or associated with nervous depression. Good examples of the chronically coated tongue are presented in the drawings (shown to the College) from a case of floating kidney and neuralgia under morphia, and one from a case of chronic dysentery under opium. The coat may be variously tinted by accidental circumstances, especially by coloring matters, which are swallowed or vomited. It may be made bright-yellow or olive-green by bilious vomit, or brown by fæcal vomit. Iron will sometimes make it inky. Iron and port wine used alternately will sometimes convey a deep brown to the coat, tannate of iron resulting from this combination. I once knew an intensely black color to be imparted to an otherwise light-colored coat by artificial teeth. Probably some mercurial amalgam had reacted with sulphuretted hydrogen in the coat and produced the black color.

The coated tongue may lose its coat in two ways: it may gradually thin off with a shelving edge, exposing a moist and natural surface, or it may break away in flakes after displaying a surface which is red and dry; the former method, implying, as it usually does, the restoration of the healthy surface, is obviously of the better omen.

Often, in circumstances which will hereafter claim attention, the coat becomes dry, brown, and cracked, sometimes presenting rectangular fissures like crocodile's skin. This amounts to what I shall presently describe as the *encrusted tongue,* towards the making of which the coated tongue is a step.

Microscopic examination of the coated tongue shows as the essential change excess of epithelium of every kind. The papillæ are prominent, their pillars of corium stand well out,

and upon them are long processes of the carmine-refusing epithelium, which is horny, superficial, and old. Between these protrusions is a superabundance of the full-bodied nucleated epithelium, such as belongs to the intervals by which these spaces are more or less filled up. In some cases this accumulation is surmounted by a thin layer which has undergone the horny transformation. When this is so, it easily accounts for the continuity of whiteness, since epithelium of this sort is, when moist, white to the naked eye. The continuity of whiteness, however, upon which the definition of the coated tongue rests, is more commonly maintained by the approximation of the long processes, and the filling of the intervals by accidental matters. There is as yet no marked injection, no great evidence of overgrowth in the Malpighian layer, no constant hypernucleation of the corium. What alteration of growth there is is in the direction of too much; but there is no such extravagance as will be noted in some conditions to be presently considered.

On and about the papillæ parasitic growths such as have been already described are often abundant, but as these are not especial to this variety of tongue, nor add very materially to the bulk of the coat, they need no detailed notice.

Clinically, the coated tongue brings with it a further increase of acute and febrile disease. Taking together the moist and dry kinds, pneumonia or pleurisy was present in ten of sixty-eight cases; typhoid was slightly more frequent than in the preceding classes; while other febrile disorders now intrude themselves into the list. Pyrexia and prostration are both on the ascent, showing that the generally coated tongue is associated with a larger proportion of constitutional affection than where the coat is more partial. It was observed that the saliva was noticeably deficient in a larger proportion of cases than heretofore—in twelve of sixty-eight of the coated tongue, in only five of sixty-nine where the coat was partial.

4. Coated White; Normally Moist.

Cerebral hæmorrhage	1
Hemiplegia (cause uncertain)	2
Pneumonia, pleuro-pneumonia	6
Pleurisy	2
Laryngitis	1
Bronchitis	2

Cancer of lung	1
Phthisis.	1
Phthisis and hæmoptysis	2
Aneurism (?)	1
Acute obstruction of bowel (small bowel)	1
Chronic obstruction of bowel (sigmoid flexure)	1
Diarrhœa	2
Ulcer of stomach (hæmatemesis)	1
Vomiting (uterine)	1
Dyspepsia	1
Repletion	1
Stricture of œsophagus	2
Starvation	1
Peritonitis	1
Chronic albuminuria	1
Uræmia, vomiting, suppression of urine	2
Acute rheumatism (including cardiac affection)	1
Sub-acute rheumatism (including cardiac affection)	3
Chronic rheumatism and sciatica	1
Typhoid	4
Febricula	1
Erysipelas	1
Erythema nodosum	1
Convalescent	2
(Total . . . . 48)	

PARTICULARS RELATING TO PRECEDING CASES.

Pyrexia (temp. 102° to 104°)	9
Hyperpyrexia (temp. over 104°)	0
No observations as to temperature	5
Much prostration	11
Prostration not severe	4
Chiefly on liquid diet	11
Strictly limited to liquid diet	15
No food by mouth; fed by rectum (stricture of œsophagus)	2
Dry diet	0
Saliva abnormally deficient	6

Average temperature of 41 cases, 99.1°.

Died, 11; recovered, 15; improved, 21; not improved, 1.

4 (D). COATED WHITE; PARTLY DRY.

Hemiplegia	1
Myelitis (acute)	1
Valvular disease and disease of aorta	2
Pneumonia	2

Phthisis and pneumonia (?)	1
Acute obstruction of bowel	1
Tonsillitis	1
Dyspepsia	2
Repletion (surfeit)	1
Chronic albuminuria	1
Diabetes	2
Acute rheumatism	5
(Total	20)

PARTICULARS RELATING TO THE ABOVE CASES.

Pyrexia (temp. 102° to 104°)	6
Hyperpyrexia (over 104°)	0
No observations as to temperature	2
Much prostration	3
Prostration not severe	0
Chiefly on liquid diet	12
Strictly limited to liquid diet	3
Dry diet	0
Saliva abnormally deficient	6

Average temperature of 18 cases, 99.5°.

Died, 6; recovered, 5; relieved, 8: uncertain, 1.

CLASS 5.—STRAWBERRY TONGUE.

This needs but a brief mention. Usually with what may be called the acute coated tongue, more especially when this approaches the plastered character to be next noticed, the fungiform papillæ show through the coat at the tip and edges. These are often more or less injected, especially with scarlatina, where they show in a striking manner the increased vascularity of the organ due to its participation in the cutaneous eruption.

The association with scarlatina would have been displayed more prominently in the table but that this disease is not, as a rule, admitted into St. George's Hospital. The strawberry state is sometimes seen in other disorders, most of which are of the acute febrile kind; pneumonia, typhoid, and perityphlitis are presented in the record. Injection of the tongue is an accompaniment of many febrile states, not only giving color to the substratum but also promoting the epithelial growth. Pyrexia is generally present with the strawberry tongue, and that to a greater degree than with the antecedent classes.

5. STRAWBERRY TONGUE; MOIST.

Typhoid	1
Scarlatina	3
Pneumonia	2
Perityphlitis	1
(Total . . . . 7)	

PARTICULARS RELATING TO THE ABOVE CASES.

Pyrexia (102° to 104°)	4
Hyperpyrexia (temp. over 104°)	0
No observations as to temperature	2
Prostration not severe	1
Chiefly on liquid diet	5
Strictly limited to liquid diet	2
Dry diet	0
Saliva abnormally deficient	3

Average temperature, 5 cases, 102°.

Died, 0; recovered, 2; improved, 3; sent out, 2.

CLASS 6.—PLASTERED TONGUE.

Plate II. No. 6.

The variety alluded to as *plastered* is well marked, has much clinical importance, and demands a separate notice. Clinically, this might be called the coated tongue of acute disease. The coat, which is white, moist, though generally not fully so, and uniform except at the edges, is spread over the dorsum, excepting a narrow margin and a small space at the tip, with a thickness, smoothness, and definite limitation which suggests the laying on of plaster. The edges, though definite, are somewhat bevelled, and are perforated as they thin with the red fungiform papillæ, after the strawberry style. Sometimes, as in the drawing displayed, taken on the fourteenth day of typhoid, the covering is so white, thick, and fine in grain as to give the idea of plaster-of-Paris or white lead smoothly spread; at other times it is coarser and less purely white, as if a fine kind of mortar had been used. The mucous surface where exposed is often redder than natural, especially when the coat is of the whiter kind. The more recent the illness the whiter the coat. When very thick, white and strongly contrasted with the red margin, the indication is of acute disease—in other words, of disease recent and severe. Were it

not recent the coat would not be so purely white; were it not severe the thickness could not have come in the time. Each day the tongue becomes less white, and gradually assumes a dirty yellowish or brownish tint. A brown color associated with dryness and cracks commonly appears when the disease is severe, of long continuance, and attended with prostration. The properties of brownness and dryness may accompany many states of tongue, and will receive further notice.

I may in a few words indicate wherein the minute characters of the plastered tongue differ from that which is commonly coated. The difference is mainly one of degree. The papillæ are now more elongated, even so much so as to resemble those of the shaggy or furred tongue. With this we have more complete filling up of the intervals, and even some overlying of the papillæ, with matters partly derived from the tongue itself as epithelium, partly parasitic and partly accidental. This condition is a great way towards the *encrusted tongue,* the chief difference being in the presence or absence of moisture. The plastered tongue when dry becomes the encrusted. In the plastered tongue the hypertrophy of the superficial epithelium, as yet uncomplicated to any great extent, attains its climax.

Clinically, this tongue never fails to arrest attention; it is the ensign of recent acute febrile disease. The chronic coated tongue is distinguishable from the plastered by its dirtier color and the less vivid apposition of white and red. Of thirty-two cases of the plastered tongue, the table shows eight of pneumonia, three of acute bronchitis, three of acute rheumatism, six of typhoid, and four of other acute febrile affections. Pyrexia was marked in eighteen cases, while the general average of temperature was 101.6°, a higher level than has yet appeared, save with the strawberry tongue, a nearly allied condition. Prostration, mostly such as belongs to the febrile state, was recorded in eight; while the increased proportion of patients on liquid diet bears testimony to the same association. The saliva was noticeably deficient in a larger proportion of instances than with any tongue yet before us, save the small strawberry class. This does not imply merely that the tongue was dryish, which might be due to the evaporation caused by the increased heat of the body, but to lessened secretion, as was in some instances ascertained with the cannula. We see a

close association with pyrexia which stimulates cell-growth, and is probably the chief agent in producing the characteristic epithelial luxuriance, the products of which are abnormally retained partly by reason of the scantiness of saliva.

6. WHITE; PLASTERED; MOIST.

Cerebral tumor	1
Pneumonia, pleuro-pneumonia	8
Bronchitis (acute)	3
Phthisis and hæmoptysis	1
Stomatitis	1
Acute obstruction of bowel (volvulus)	1
Dysentery	2
Perityphlitis	1
Jaundice (enlargement of liver)	1
Acute rheumatism (including pericarditis, etc.)	3
Typhoid	5
Febricula (or febrile attack of uncertain nature)	1
Measles	1
Mumps	1
Pyæmia	2
(Total	32)

PARTICULARS RELATING TO THE ABOVE CASES.

Pyrexia (temp. 102° to 104°)	15
Hyperpyrexia (over 104°)	3
No observations as to temperature	5
Much prostration	5
Prostration not severe	3
Chiefly on liquid diet	16
Strictly limited to liquid diet	8
Dry diet	0
Saliva abnormally deficient	13

Average temperature of 27 cases, 101.6°.
Died, 10; recovered, 11; improved, 11.

Before proceeding to other varieties of tongues, I will, now that we have reached in the plastered tongue the climax of simple coating, look back upon the several grades of that condition, and try to collect the instruction which flows from them.

First pathologically. The most important item is the lengthening of the papillæ; and the first question, how far this depends on disuse from want of food, etc., and how far on overgrowth. Want of wear must have some effect in allowing this elongation, but it would seem to be too rapidly produced to be wholly thus accounted for. A greater length is

attained within the short duration of an acute disease than in the longer time of a chronic one, even though it entail more complete disuse. Not only are the epithelial ends to which only the wear applies elongated, but so in many cases are the deeper parts of the column which are unexposed. The epithelium also between the papillæ, which, as being less exposed than they are, must be less influenced by wear, is also in many cases increased in thickness; when it is so it gives strong evidence of over-production as against deficient removal. Coating, therefore, is the result in part of disuse, want of rubbing and washing, as the somewhat increased surface parasites show, but chiefly of morbid overgrowth.

The clinical circumstances give similar evidence. It is true that the greater degrees of coating occur chiefly in patients who are kept without much solid food. On the other hand, in many conditions in which solid food and mastication are almost completely absent, the thick and rapid covering of acute disease does not present itself. I have watched with interest from this point of view cases of stricture of the œsophagus. In three such only the lower degrees of coating were presented, though in all solids were impossible, and in two food was introduced chiefly by the bowel. In a fourth the tongue was dry and furred, but the furring came on with dryness, on which it was apparently dependent. I shall revert to the influence of diet upon the tongue, and simply say here that a comparison between the thinly or partially coated tongue of simple abstinence, and the thickly and generally coated tongue where a less degree of abstinence is associated with a febrile state, shows conclusively, as it appears to me, that in the latter condition we have something to look for beyond inaction.

I shall refer later to the effects upon the tongue of the limitation of liquid, but it is enough for the present to say that want neither of food nor of drink avails to produce the thick coating which in other circumstances is so characteristic.

A condition which may be briefly referred to in connection with coating of the tongue is diminution of saliva, though I shall consider this subsequently on a wider basis. As a general rule, whatever be the reason, the saliva lessens as the coat thickens. Taking together the lesser degrees of coat presented with the stippled, and stippled and coated classes,

this secretion was notably deficient in twelve of 170 cases. Taking together the higher degrees of coat in the strawberry and plastered kinds, the saliva was notably deficient in sixteen of thirty-nine. Thus generally up to this point, the less saliva the more coat. But it will appear later that almost total absence of this fluid may concur with a tongue which is almost absolutely without coat. For the present it is enough to point out that the deficiency does not alone account for the excess of covering under consideration.

To account for this there remain but two courses which present themselves as probable: the hypothetical existence of a morbid poison as in fevers, and pyrexia. The existence of a morbid poison as a cause of coating of the tongue is suggested by the specific febrile character of many of the cases where it is well marked; we may not be able to explain any direct connection between poison and coat, but must not on that account too hastily assume that there is none. That there is an indirect connection through pyrexia will become evident. To come to details. looking at the high degrees of coat in the classes of strawberry and plastered, I find that of thirty-nine such cases fourteen were of typhoid, febricula, scarlatina, measles, mumps, or pyæmia, and three of acute rheumatism. Thus, unless we include pneumonia, of which there were ten cases, less than half fell within the hypothesis of poison. Among the rest were many cases of local or organic disease, which were clearly outside this supposition. Among these were three cases of acute bronchitis, one of phthisis, two of perityphlitis, one of volvulus—enough to show that local disease, or disease not connected, so far as we know, with any blood-contamination, may give rise to what may be called acute coating of the tongue.

Pyrexia remains to be considered. When the tongue is normal, so, as a rule, is the temperature. I have already referred to one case as exceptional in which the tongue remained clean in the presence of pneumonia. Putting this aside, I have already referred to twenty-two cases in which, in different circumstances, the tongue was reasonably clean, the temperature was raised only once, and then only to 101.5°. Taking, now, the degrees of coating in ascending order, we find that as the coat increases so with almost exact correspondence does the temperature.

RELATIONS OF BODY HEAT TO COATING OF TONGUE.

CLASS OF TONGUE	PERCENTAGE WITH PYREXIA	AVERAGE TEMPERATURE.
Stippled	4.9	98.5°
Stippled and coated	8.6	98.7
Coated, moist	18.7	99.1
Coated, dry	30.0	99.5
Strawberry	57.1	102.0
Plastered	53.1	101.6

The annexed statement needs no explanation; whether we look at the frequency of pyrexia in the class, or at the average temperature of all cases in it, the figures are equally significant. Taking the strawberry and the plastered tongues as practically the same, the proportion between coat and heat is maintained with remarkable exactness. And we have as yet failed to find any other condition besides heat of body which has so uniform a relation to the alteration in question. That heat up to a certain point promotes the growth of cells is a fact well known to experimental physiologists. I take it on the authority of Dr. Delépine that a temperature of between 100° and 104° is favorable to the growth of tissues, one of above 105° detrimental to it. It probably very rarely happens that the dorsum of the tongue, exposed as it is, and necessarily cooler than the constantly covered parts, reaches a temperature high enough to lessen growth; almost any degree of elevation possible to the tongue in our present surroundings must be within the limits of that which tends to increase. In pyrexia, therefore, we have, if not the only maker of coat, certainly the chief one.

CLASS 7.—THE FURRED OR SHAGGY TONGUE.

Plate II. No. 7 D.

As the papillæ of the coated tongue continue to lengthen, and adventitious matter to collect between and upon them, the advancing process presents itself in two shapes, which differ as the elongation or the accumulation preponderate. When there is great projection of the papillæ, so that these stand out distinctly, the terms *furred* or *shaggy* represent the condition. When these shapes are covered in, and levelled

over by matter, whether derived from the tongue itself, or from without, the term *encrusted* may be employed. The two conditions occur in many similar circumstances, and are continually intermingled; nevertheless, they occur separately, and must be so described. A stippled or dotted tongue by increase of each point of coating may become furred, a process which dryness facilitates. In a different way a thickly coated tongue may become furred. The lengthened papillæ may present themselves as a coarse pile-like plush, and so make the *moist furred tongue.* A little drying will cause the threads to collect into sheaves, and so form the *dry furred tongue* which I am about to describe.

To the naked eye and during life the fore part is irregularly rough, with large pointed papillæ, on the tips of which are brown spots, or the whole may be more or less brown. The central and back parts are often covered with irregular pointed masses, such as are represented in the drawing (exhibited), frequently more or less mixed up with and obscured by dry crusts, often like crocodile's skin. I have seen after death (in a case of malignant disease of the larynx) much of the hinder region of the tongue covered with a shaggy villous growth, like coarse hair; but in life this hirsute state is commonly much obscured by incrustation.

Under the microscope the chief characteristics of the tongue are the enormously elongated filiform papillæ, every part of which is increased, both the deeper portion derived from the corium and the superficial epithelial part, the latter most so. Micrococci are sometimes present on the tips of the papillæ, but do not add materially to their bulk.

The elongation may conceivably be due either to overgrowth or deficient removal; there is often some increase of the deep epithelium, but seldom much; occasionally some over-nucleation of the corium is present. In fine, there is some evidence of hyperplasia, but not enough to account for the striking changes which the tongue presents. These would seem to be mainly due to want of wear, of which the papillary ends, hardened by dryness, are especially resistant. Beyond the resistance which dryness entails, it is possible that the unnatural state of surface which goes with it may act as an irritant and stimulate growth.

As to the clinical relations of this tongue, I have been able

to collect, notwithstanding the generality of dryness, a few instances in which the furred tongue was moist, or at least not dry. One of these was of a kind sometimes seen where great elongation of the papillæ is consistent with appetite and health. The only conditions which could be suggested as provocative of the state of tongue in this case were old age and constipation. I have referred to the state of pile, which may be regarded either as an advanced stage of the coated tongue or an early one of the furred. In the table it is seen to be associated with pneumonia and with anæmia.

The *dry* furred tongue is of more importance than the moist, but it occurs in different circumstances and is by no means uniform in its indications. It may succeed, as has been shown, either upon the dotted tongue or the coated, in the course of advancing disease; it may also occur with retrogressive disease, as when the encrusted tongue sheds its coat so as to expose the subjacent elongated papillæ. The class therefore presents much clinical variety. It has been shown that the furred or shaggy tongue is largely the result of disuse and want of moisture, which latter condition is nearly essential, though not absolutely so. The papillæ harden with dryness, as has been stated, and become abnormally resistant of friction, which, in the absence of solid food and mastication, is diminished. The saliva is obviously deficient, as judged by the difficulty of spitting and by the results of catheterization of the parotid. It must be stated, however, that the latter operation was not often performed, the extreme illness of the patient often opposing a hinderance. The fact, however, was clear without this test; and the conclusion warranted that the dryness was in general due to want of this secretion.

The accompanying tabulation speaks for itself. I need not allude again to the instance where a profuse hirsute growth was conjoined with sarcoma of the epiglottis and tonsil; here the results of want of friction were chiefly evident, though there was some evidence of overgrowth in hypernucleation. I may next refer to a group of cases in which, as presumably in the last case, the condition was present with little or no pyrexia. This includes two cases of coma from brain-disease, two of advanced cirrhosis, one of enlarged liver of uncertain nature, two of diarrhœa, and three of enforced dry diet. Thus dehydration, be its cause what it may, is a definite factor in

producing the tongue in question. In other cases—to wit, typhoid and acute rheumatism—pyrexia was present, but it is clear that this is not essential. It is worth observing how infrequent is pyrexia, and how low its range in the dry tongue now in question, as compared with the plastered moist tongue last discussed. This shows how little of the desiccation is to be attributed to evaporation connected with increased heat of body. And I may make a similar negation with regard to another condition to which dryness and furring have been attributed—namely, habitual openness of the mouth, especially during sleep; I find but three instances in which this attracted notice, and I am persuaded that other causes of drying and furring are far more important.

In short, the dry furred tongue is essentially due to two causes which are connected together—want of saliva and want of wear. Among the causes of want of saliva, the most important is a state of system which cannot be otherwise defined than as failure of nutrition and vital power. With how great a variety of disorders this failure is associated I need not recapitulate; I have expressed the greater degrees of it by the term "prostration."

7. Furred or Shaggy; not Dry.

Pneumonia, pleuro-pneumonia	2
Perityphlitis, perforation of appendix	1
Chronic constipation	1
Granular kidney; uræmia	1
(Total . . . . 5)	

Observations Relating to Preceding Cases.

Pyrexia (temp. 102° to 104°)	1
Hyperpyrexia (temp. over 104°)	0
No observations as to temperature	2
Average temperature (of three cases)	99.8°
Much prostration	2
Prostration not severe	0
Chiefly on liquid diet	2
Strictly limited to liquid diet	1
Saliva abnormally deficient	2

Died, 3; recovered, 1; not improved, 2.

7 (D). Furred or Shaggy; Dry.

Disease of brain (coma)	2
Hemiplegia (embolic)	1
Chorea	1

Valvular disease	1
Aneurism or dilatation of aorta (dry diet)	3
Broncho-pneumonia	1
Pleurisy	1
Cirrhosis of liver	2
Enlargement of liver (cause uncertain)	1
Stricture of œsophagus	1
Diarrhœa	2
Perityphlitis	1
Abscess (in axilla)	1
Acute rheumatism	1
Typhoid	1
Convalescence from typhoid	1
Febricula	1
Movable kidney and rheumatism	1
Chyluria	1
(Total . . . . 24)	

PARTICULARS RELATING TO PRECEDING CASES.

Pyrexia (temp. 102° to 104°)	3
Hyperpyrexia (temp. over 104°)	0
No observation on temperature	2
Average temperature of 22 cases	99·1°
Much prostration	6
Prostration not severe	2
Chiefly on liquid diet	9
Strictly limited to liquid diet	5
No food by mouth (fed by rectum)	1
Dry diet	3
Saliva abnormally deficient	8 ?

Died, 9; recovered, 7; relieved, 8

LECTURE II.

Class 8.—Encrusted Dry and Brown Tongue.

Plate III. No. 8.

Mr. President and Gentlemen,—The variety about to be described is distinct enough, though it has points of contact with the two preceding classes, as a later stage of which it presents itself. The essential characteristic is the covering of the tongue with a brown dry crust, which surmounts and conceals the papillæ. It occurs as a later stage of the furred tongue, the spaces between the elongated papillæ getting filled up, as it would seem almost necessarily, and the accumulation carried up so as to level or overtop their summits. It occurs also as a later stage of the coated tongue, more particularly of that kind which has been described as plastered, the growth of the papillæ and of the epithelium in the intervals and the accumulation of parasites on the dry surface producing the result.

To the naked eye the characters of the encrusted brown and dry tongue are almost sufficiently described by these three words. It is irregularly covered with brown dry incrustation, variously broken and fissured. Sometimes the cracks are quite irregular; often the deeper are transverse, with longitudinal longer and shallower fissures, which together divide the crust into more or less rectangular scales. The surface, particularly in front, often displays, instead of incrustation, coarse elongated papillæ which are antecedent to it, while towards the back and central parts the coat covers and conceals these prominences. The color is generally brownish and the upper surface rough and dry, while the under part is smooth, uncoated, and only clammy. The teeth are often encrusted with brown matter.

It may be asked why is the encrusted tongue brown? Firstly, because it is dry. Many animal substances dry brown, among others the white coat of the tongue. I have dried a thickly coated white tongue at the temperature of 100°, and

also at that of an ordinary room. When dry it became brown, but not completely so. A brown, dry, encrusted tongue when soaked in water became nearly white, but not quite, and brown as before when re-dried. Thus dryness appears to be in part a producer of brownness, but staining by food and medicine no doubt helps. Many colored matters—beef tea, wine, etc.—go into the mouth of a sick man, and cannot but discolor the tongue if the natural wash by saliva is wanting. The crusts on the teeth are probably similarly produced. They are not due to the drying of saliva on the teeth, for saliva at the heat of the body dries white.

With the microscope the chief points are the elongation of the papillæ, and their being embedded and overlaid by a heterogeneous brown mass, which will be further noticed. The incrustation differs from what has been shown to form the coat or fur, though attached to it so as to be inseparable without fracture. Traced in section from within outwards, it presents first much epithelium, more or less horizontally arranged, flattened, but usually not old enough to have lost the power of taking carmine. Through this project long papillæ, chiefly consisting of horny, untinting epithelium, upon the exalted summits of which rests a confused mass which constitutes the crust. While the tops of the papillæ support this material, their intervals are filled up by it. Thus there are two constituents of the covering of the encrusted tongue: first, a large growth or accumulation of epithelium, much of which is in the shape of elongated papillæ; and, secondly, a less definite structure, which lies upon this and in its interspaces. The superimposed substance is mixed and irregular; much of it can be described only as amorphous; in it can be distinguished epithelial cells disposed without regularity, fat-globules, occasionally other adventitious and accidental matters, and last, not least, a multifarious collection of organisms, chiefly vegetable. In two cases, in one during life in the other after death, I found lying on the tongue large detached masses of dry brown crust, one of which was not less than a quarter of an inch thick. Sections of these prepared in different ways were examined; I show a diagram representing one of these sections made from a drawing which I owe to the skillful hand of Dr. Delépine. Both masses showed a profusion of vegetable organisms, chiefly micrococci and the *Oïdium albicans.* I am

indebted to Dr. Delépine for a minute description of these organisms, which I insert below.[1] Besides these, there were many

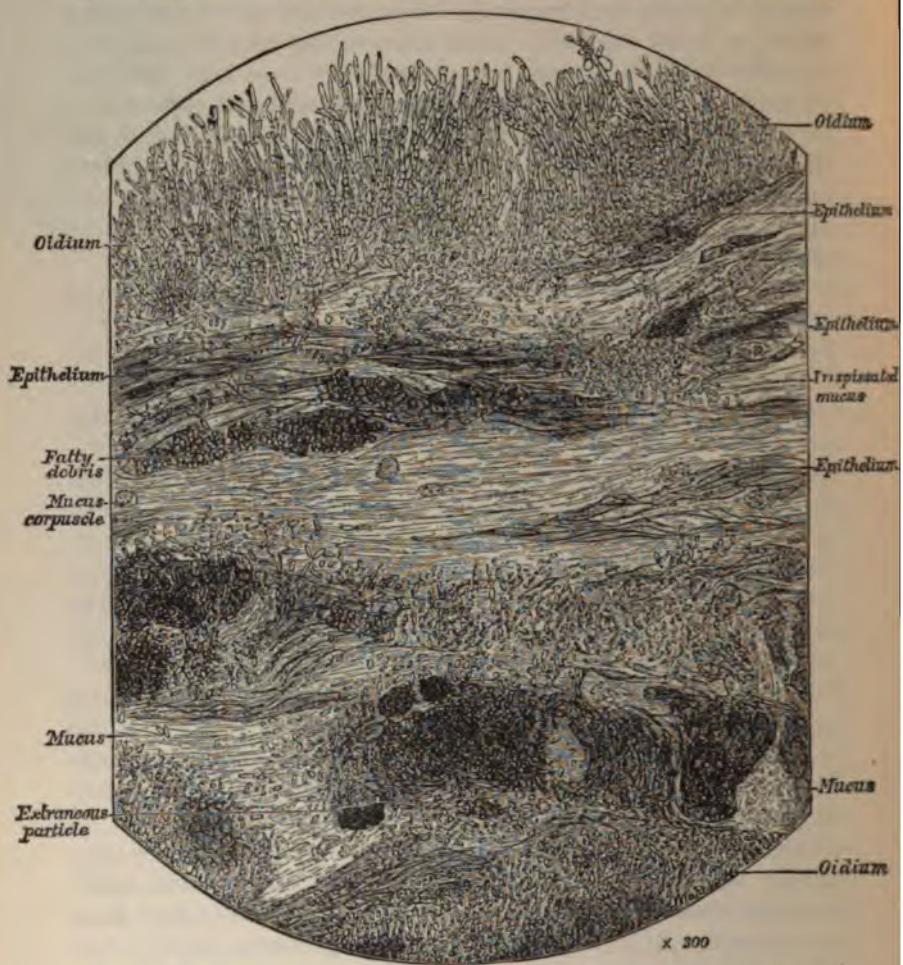


FIG. 1.—Transverse section of flake of coat spontaneously detatched from dry encrusted tongue from a man who died of sarcoma of the lung under the care of Dr. Cavafy. He was about a week dying, with open mouth and muttering unconsciousness. The flake was found after death; it was about a quarter of an inch thick, and looked like a cinder.

[1] The organisms found in the fur belong chiefly to the two following forms:

1. Masses of micrococci.
2. Branching septate filaments, ending in large spore-like cells.

The second form of organism must be referred to the class of Hypho-

strands of horny or superficial epithelium, many epithelial cells of the deeper kinds, many fat-globules, and much indeterminate matter. The dry incrustation differs from the white moist coat chiefly in this: whereas the major bulk of the white coat is epithelial, a very large proportion of the dark dry crust is parasitic. In the deeper parts of the epithelial structure, more especially in the Malpighian layer, there is often much

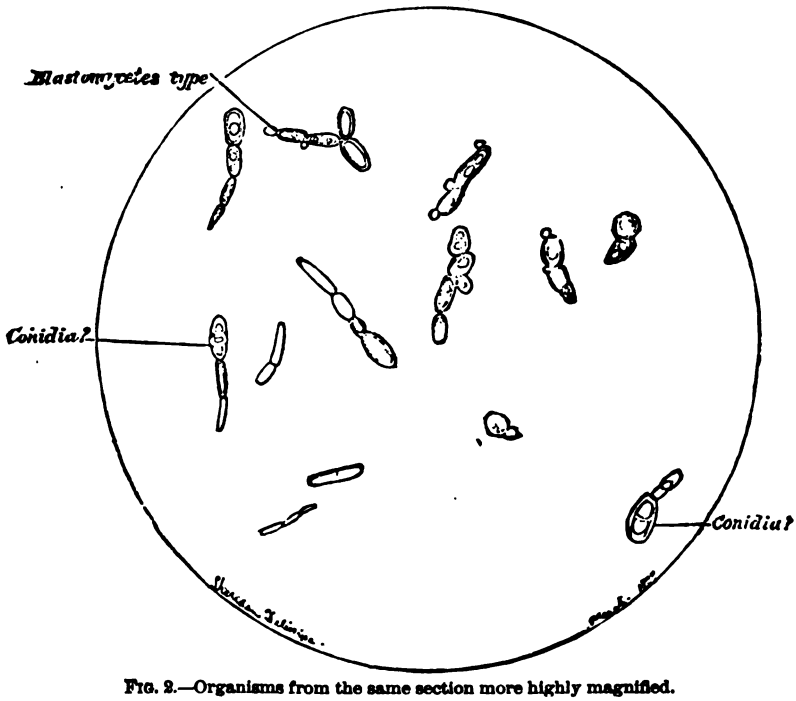


FIG. 2.—Organisms from the same section more highly magnified.

profusion of nucleation or cell-growth, the corium is in many cases distinctly hypernucleated and often over-injected. More

mycetes fungi, and probably to the species or variety called *Oïdium albicans* by Robin. A great portion of the growth, however, has all the characteristics of one of the Blastomycetes; this, however, is not surprising when the views of Grawitz, Cienkowski, Naegeli and others are kept in mind. Grawitz, for instance, says that *Oïdium albicans* is nothing else than a modified yeast (*Mycoderma vini*); Naegeli maintains that there is no difference between the genera Torula and Mycoderma.

It may be also remembered that spores like those observed in this case were said formerly to belong to an alga (spurious), the so-called *Cryptococcus cerevisiæ*.

rarely leucocytes are extruded within the papillæ and elsewhere.

To sum up the evidence afforded by the morbid anatomy of the encrusted dry tongue, we see first the results of the absence of friction and irrigation, most conspicuously in the accumulation which forms the crust. It is probable, also, that the elongation of the papillæ may be partly attributed to this cause. But there are also signs of increased cell-formation and nutrient activity, so that we witness here a double process, over-production conjoined in larger measure with deficient removal. It is obvious that the dryness which helps to define this tongue has played an important part in it, both by want of scour and also by induration.

Coming to clinical considerations, much that has been said about the furred tongue applies also here. It will be convenient to take together two classes which, though I have separated them for some of the purposes of the inquiry, are not to be distinguished save as the earlier and later stages of the same state. I shall for clinical purposes fuse together the tongue which is completely encrusted and that which is in the process of shedding its crust in the way to become denuded. So various are the conditions under which the encrusted tongue is seen that it is not easy to discern what the link of association may be, except the somewhat loose one of severe illness, and that of some standing. The instinctive sense of the physician carries him thus far. His looks are ominous, he talks of the "typhoid state," and he orders stimulants.

Analysing the details in the two tables before us (Nos. 8 and 9, see pages 635 and 639), comprising together thirty-five cases, pneumonia takes the first place in frequency with six cases; then come pyæmia with four and continued fever with three. Acute rheumatism with hyperpyrexia occurred in one instance, and erysipelas in one. In short, there is scarcely any protracted and depressing febrile disorder in which, if I may supplement the scanty records before us with unrecorded experience, this tongue is not apt to be developed. It is often characteristic of typhus by means of the black color it then presents—possibly due to hæmorrhage into the coat (but of this I cannot speak from observation). The encrusted tongue is so commonly looked for in advanced typhoid that it is often spoken of as the "typhoid tongue," though by no means the

only variety displayed in this disease. But the association with fever does not contain the whole story, nor is the febrile condition essential. This tongue may concur with its complete absence. The abstract shows that of the thirty-four cases in question fifteen displayed a temperature above 102°, eight a temperature under 99°, in three of which it was subnormal. The low temperature was manifested with abdominal cancer, advanced phthisis, albuminuria, syphilitic mania, diarrhœa, alcoholism, and prostration from an unknown cause. Thus pyrexia, though often present with the encrusted tongue, and no doubt by the evaporation which it entails helpful to its production, is by no means necessary to it. The average temperature is lower than with the plastered tongue. It is scarcely needful to inquire, after what has been said, whether this tongue is to be generally attributed to openness of the mouth during sleep or coma. The dry encrusted or furred tongue—for the two have similar clinical relations—is an especial property of coma or insensibility. I have not recorded any case in which well-marked coma persisted for more than twenty-four hours without one or the other being developed. The records are scanty, but a larger experience supports the general rule that with coma or long unconsciousness the tongue becomes as described. Many reasons are obvious—dryness and disuse the chief. But why the dryness? It is manifest that coma, if it involve habitual openness of the mouth, must tend to dry the tongue; but if health is good in general respects, it does not appear that the mere passage of air over the tongue in respiration can produce anything like the degree of desiccation in question. I have noticed how little dryness has resulted in a person otherwise well whose nostrils have been surgically plugged, and we must take another point of view. Coma, however it acts, is connected with only a minority of cases in which the tongue becomes dry and encrusted. Putting aside all states of coma and unconsciousness, all in which the mouth has been habitually open from other causes, and all states of pyrexia, there remain many conditions, and those most various, in which this tongue has presented itself. Advanced phthisis, intra-thoracic and abdominal cancer, idiopathic anæmia, and diarrhœa have been mentioned; all were attended with much depression or prostration, and, indeed, it appears that, whatever else be present, a condition of much

illness or lowered vitality runs through all and supplies the common factor. It has been shown that a similar condition of prostration often attends the furred tongue. I should have preferred to have been more explicit, but it is not easy to narrow the statement within a more strict definition. To most of the conditions the term "sinking" would be applied as indicating what is present or threatened. There is not necessarily any loss of consciousness nor any obvious failure of the nervous system. Sense and senses may be alike unimpaired. There is, as a rule, failure of circulation, of muscular strength, and of nutrition. Perhaps the term "asthenia" best implies the state. Any disturbance may be superadded, but none other is generally essential. I say *generally*, for direct dehydration will cause the state of tongue in question. This is seen in the effects of dry diet, and perhaps less simply in the effects of diarrhœa. It is to be observed that exhaustion by suppuration, as will be seen, causes other changes in the tongue than this.

It has been shown that the essential local change is dryness, and that this, as a rule, is not due to direct dehydration or to increased evaporation from pyrexia or patency of the mouth. That it is due to deficient secretion of saliva is a necessary conclusion, warranted by the obvious deficiency of liquid in the mouth, by the difficulty or impossibility of spitting, and by the usually fruitless result of catheterization of the parotid, notwithstanding that acetic acid may be applied to the tongue as a stimulant to the gland. I shall revert to this point, but am not wrong so far in assuming the dryness to be, as a rule, due to suppression of this secretion. Associating this with the asthenia which accompanies it, we cannot but regard the local as the result of the constitutional state, and probably may accept it as a sign that the prostration is telling upon the functions of at least some of the organs. The bowels, kidneys, and skin do not obviously participate in the failure; but how about the gastric and other digestive juices? Observations here are difficult or impossible, but it may be conjectured that the salivary failure does not stand alone, and we may accept the state of tongue before us as an index not only of asthenia, but of a failure of certain vital functions connected with nutrition.

8. Encrusted, Dry, and Brown.

Pericarditis, hydrothorax, tapping often	1
Pneumonia	2
Phthisis	1
Cancer of lung	1
Block in rectum and hepatic ascites	1
Choleraic diarrhœa	1
Granular kidney and uræmia	1
Uræmic coma	1
Diabetic coma	1
Obstructive jaundice	1
Cancer of pancreas, etc.	1
Acute rheumatism	1
Idiopathic anæmia	1
Typhoid	1
Typhus	1
Erysipelas	1
Pyæmia	3
Rapid emaciation, cause uncertain	1
Alcoholism	1
(Total	22)

Particulars as to the Foregoing Cases.

Pyrexia (temp. 102° to 104°)	8
Hyperpyrexia (temp. over 104°)	1
No observations as to temperature	1
Average temperature of 21 cases	100.3°
Much prostration	12
Prostration not severe	—
Chiefly on liquid diet	13
Strictly limited to liquid diet	—
Dry diet	0
Saliva abnormally deficient[1]	8

Died, 14; recovered, 7; relieved, 1

Class 9.—The Process by which the Coated, Furred, or Encrusted Tongue becomes Red, Smooth, and Dry.

Plate III. Nos. 9 *and* 10.
Plate IV. No. 11.

The cleaning of convalescence requires no further notice; the shelving off of the coat at the tip and edges has been de-

[1] The extreme illness of many of these patients made it difficult to estimate the absence of saliva by attempts to spit or by catheterization of the duct. The number therefore appears small. It is probable that saliva was wanting in nearly all, so far as the state of the mouth could be taken as an index.

scribed. In less favorable circumstances the process of making bare occurs in this wise: The incrustation is very dry and correspondingly brittle, and the epithelium beneath it, in connection probably with the same want of moisture, does not grow properly. The coat or crust now wears off more or less at the tip and edges, in a gradual manner, and displays not the normal surface, but a red and dry one, usually covered with a delicate membrane, so thin as possibly to escape notice, but discernible to a careful eye. Under the microscope it is evident enough. After, or more or less together with, the exposure of the fore part of the tongue, the central part loses its covering along a broad stripe which reaches from this point not quite to the back. This may be an inch or rather more in width, and is the part of the tongue most exposed to the breath, and consequently the driest. The clearing is often effected by a very obvious cracking and breaking away of the crust, which may be so rapid that I have seen a much-encrusted tongue become nearly naked in a day. The exposed surface may look raw, but is seldom absolutely so, for it is skinned over with the dry translucent membrane which has been described. This stretches straight along the surface, like the arachnoid of the brain, not dipping between the papillæ, the outlines of which it obscures. The bare and polished stripe is often fringed with white fur or coating as a line down each side, outside which is the nearly normal lateral margin. After a time the whole upper surface becomes nearly equally red, dry, and bare, the surface being intersected with fine or deeper lines or cracks, some longitudinal, the deeper transverse. The coating is retained longest at the root. A resumption of moisture is a sign of constitutional improvement, and precedes a gradual reclothing—too seldom observed—which restores the tongue to its normal state.

I will now bring the microscope to bear upon the process which has been described. I pass over the gradual change of convalescence to consider the modes by which the morbidly dirty tongue becomes morbidly clean. Starting with the elongated papillæ of the furred tongue, these, together with all the superficial epithelium, are often swept off accurately down to the Malpighian layer, which retains its place, and still uniformly clothes the corium. In time, or almost simultaneously, this may become covered afresh with epithelium of

the horny sort, not at first as in health, but in the shape of the thin membrane of which I have spoken. Supposing the denuding process to continue, the Malpighian layer itself is removed and the corium exposed, making a surface which is uneven microscopically, though to the naked eye smooth, and necessarily red and raw. This process is attended with vascular injection, hypernucleation, and often the extrusion of leucocytes. Much of the injection and hypernucleation may be the consequence of the loss of the protective epithelium, with exposure and irritation of parts which should be covered. But in some cases it is apparent that a general inflammatory infiltration of the superficial parts precedes the denudation, and helps to produce it by a destructive process. This is most marked with scarlatina, where the infiltration is often most abundant. This may be due to the especial effect of the disease, which the tongue appears to share with the skin and throat. I have now traced the flaying operation down to the true skin. All that is special to the tongue has gone, at least in places; but more often, as I have said, the process stops on reaching the Malpighian layer, and here begins a process of repair to which I have alluded, but to which I must revert. The tongue is now red, level, and polished. To the hasty eye it looks like raw beef—a similitude often used; but the beef is wrapped in silver paper. A continuous layer of horny epithelium of extreme tenuity presents itself, and, resting on the more elevated remnants of what were papillæ, stretches from one to another without dipping into the hollows, like the roadway of a bridge. The hollows, however, are not empty, but contain epithelium of the deeper sort. The somewhat gradual process which I have described is sometimes replaced by one into which violence more obviously enters. This relates especially to the encrusted tongue. The felted mass of crust breaks through its deeper part, the fracture passing through the bases of the papillæ, leaving their broken stumps standing. These quickly become levelled down, and the Malpighian layer soon presents itself with the delicate membrane, as described. In this breaking away of the crust, accident cannot be excluded, but more is probably due to the rising up of new epithelium from beneath, by which the brittle, and mostly effete, mass above is pushed from its stool. Probably

something of this sort happens when scarlet-fever patients and snakes shed their skin.

It is no part of my purpose to treat of the special diseases of the tongue, but one of them bears instructively on the question before us in presenting a mode by which the coat and the papillæ themselves are removed. Dr. Cavafy gave me the opportunity of examining the tongue after rapid and great swelling in a condition of glossitis or acute inflammatory

FIG. 3.—Acute œdema of the tongue connected with albuminuria. The papillæ are in process of detachment by the disproportionate swelling of the parts beneath. The exposed part has acquired the level membrane, such as is represented on Plate IV.

œdema, which had come on in the termination of albuminuria. The state was clearly inflammatory, not dropsical. The swollen tongue, which I did not see during life, presented, when examined in section, an extensive region on its upper surface, which was so far denuded that the corium was covered only with a thin straight membrane, such as has been elsewhere described. The membrane in this case, however, was different from what is usually seen in being carried along an uninterrupted surface without having to bridge any depressions or

sulci. This appeared to be due to the stretching of the surface by swelling, all the notches between the papillæ having been pulled straight and the serrated surface converted by elongation into a level one. At the edges of this level plain were upstanding papillæ, those nearest to the smooth surface showing the process of breakage by which it had become denuded. The surface had apparently been made bare by the detachment of the papillæ in the process of stretching. How this stretch-

FIG. 4.—Portion of the muscular structure of the same more highly magnified, showing the interstitial exudation which was the chief cause of the swelling.

ing had been produced was made evident by an examination of the deeper parts of the tongue, where a large granular exudation was irregularly disposed between the muscular fibres. I give sketches showing the levelled surface, the adjoining partially detached papillæ, and the interstitial intrusion to which the swelling was due.

9. FURRED OR ENCRUSTED, BECOMING DENUDED; GENERALLY DRY.

Tetanus	1
Syphilitic mania	1
Valvular disease	1
Pneumonia	4

Cirrhosis	1
Dysentery, abscess of liver	1
Cancer of pancreas, etc.	1
Peritonitis, fæcal extravasation	1
Typhoid	1
Pyæmia	1
(Total . . . . . 13)	

PARTICULARS RELATING TO PRECEDING CASES.

Pyrexia (temp. 102° to 104°)	5
Hyperpyrexia (over 104°)	1
No observations as to temperature	3
Average temperature in 10 cases	100.8°
Much prostration	7
Prostration not severe	—
Chiefly on liquid diet	9
Strictly limited to liquid diet	2
No food by mouth (fed by rectum)	0
Dry diet	0
Saliva observed as deficient[1]	2 (?)

Died, 10; recovered, 1; relieved, 1; not relieved, 1

CLASSES 10 AND 11.—BARE, RED, AND DRY TONGUE.

Plate III. Nos. 9 *and* 10.

Plate IV. No. 11.

In the description of the process of denudation I have anticipated much that applies to the bare tongue. The completely bare tongue, red, raw, and dry—for this state does not seem to be attained except with dryness—is comparatively rare. The term *completely* bare or denuded relates to the completeness of the loss of the epithelium in certain places, not to its spread over the whole tongue. This change is more limited in extent than the more shallow divestures which have been described. I have already traced the process down to the Malpighian layer, which has become covered anew with a straight membrane instead of the normal serrated one. Should repair be exceptionally wanting, this membrane is not formed, but the Malpighian layer is left to break away and lay bare the corium beneath. Occasionally it would seem that the destruction occurs after the formation of this membrane, which

[1] Mouth invariably dry, but special observations on saliva not generally made, or practicable.

it involves, for the broken ends are often seen overhanging a tract of excoriation. The exposure is effected not quite abruptly; the neighboring epithelial structures shelve off until the patch of complete exposure is reached. Here no epithelium of any kind remains; the corium is laid bare to the cavity of the mouth, and is even sometimes itself encroached upon. I have not seen this process descend quite to, though sometimes it has approached, the muscular fibres. The exposed corium sometimes shows little change of structure, more often it is infiltrated with leucocytes, sometimes to such an extent that the surface appears to be entirely composed of them. There is also injection of the blood-vessels. The infiltration is probably generally due to irritation by the unaccustomed contact of the contents of the mouth, and is subsequent, not antecedent, to the loss of covering. Even in scarlatina, where the inflammatory exudation may possibly be a part of the eruption, the nucleation is greater in the exposed parts than elsewhere.

For clinical purposes I shall place together two classes that cannot be always distinguished except with the microscope, and give a common consideration to the red denuded tongue, whether absolutely bare or protected only by the thin membrane which has been described.

The qualities of redness, smoothness, and dryness are nearly related. If the tongue be uncovered by the opaque superficial epithelium it will necessarily display the vascularity of the deeper parts; and this is usually increased because the tongue is irritated by exposure. The denuded tongue is dry partly because the loss of epithelial protection allows of increased evaporation from its surface, but chiefly from want of saliva. Want of saliva may encrust the tongue; it may also make it bare: of this more anon. This variety of tongue is that on which aphthous growths are most apt to occur.[1] It presents

[1] It is worth remarking that, though aphthæ are noticeable chiefly on the red smooth tongue, upon the otherwise clean surface of which their white forms are conspicuous, yet the special vegetation of which they consist is found in other circumstances, being a constituent of crust, as already shown. Probably the *Oïdium albicans* tends to gather upon an inflamed, ulcerated, or otherwise altered surface rather than a healthy one; and, like other parasitic growths, is apt to present itself in conditions of depression which are also those in which the red, raw tongue is developed. What may be the connections between the *oïdium* and

itself in diverse circumstances, generally of exhaustion. The most prominent fact is its occurrence as the result of exhaustive discharges, especially of pus, and it is linked with the constitutional state described as hectic. The Table shows that of thirty cases seven were connected with an ostensible escape of pus, including one case of lumbar abscess, one of empyema, two of discharge of pus with the urine, one of dysentery with open hepatic abscess, and two of advanced phthisis with much expectoration and diarrhœa. Besides these, in which loss of pus was obvious, there was one instance of profuse dysenteric flux, in which an abscess had formed in the liver but not found exit; two cases of lardaceous disease with the customary discharges, and five of advanced diabetes, brought to fifteen, of thirty, the number of instances in which this state of tongue was associated with exhausting discharges. The frequency of diarrhœa deserves remark. It has not been shown so far that simple diarrhœa causes it, but diarrhœa was prominent with other morbid conditions, notably dysentery and phthisis. The most striking tongue of this kind which I ever saw (it was as red and almost as smooth as sealing-wax) was in the case already referred to of acute dysentery, with unopened hepatic abscess.

I have to thank Sir Joseph Fayrer for giving me some of his Indian experience, with regard more especially to tropical diarrhœa. In advanced cases of this, the tongue, he says, is shrunken, red, polished, and smooth; the papillæ have disappeared, and the epithelium is stripped off in patches. The gums, lips, and buccal mucous membrane are often aphthous. The tongue is very tender; alcohol and salt cause great pain in passing over it. As significant signs of convalescence, Sir Joseph notices a softening of the tongue, a change of the red to pink, loss of tenderness, and the reappearance of the papillæ.

There remain cases in which the red, dry, smooth tongue has presented itself without any obvious association with discharge. Among them were five of pneumonia, all with much depression; one was double, and attended with maniacal de-

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the state of surface which this tongue affords, and what in further detail are the relations of thrush to the general health, are matters upon which I had hoped to have said more, but have not been fortunate in obtaining tongues thus affected. The subject is well worth further attention.

lirium; one was complicated with delirium tremens; another was in a drunkard; two were attended with great prostration (not explained). There were two cases of typhoid, of which one was characterized by sleeplessness and nervous agitation; one of tubercular peritonitis, one of biliary colic, and one of a dangerous gorge of grapes,—all these conditions being attended with much depression. Advanced tubercular disease presents itself as a not infrequent associate of this tongue; there were five cases of this sort in the thirty. Pyrexia is not needful, though often present. A temperature not above normal was found with it in cases of lumbar abscess, pyelitis, lardaceous disease, several of diabetes, and some under dry diet. As compared with the encrusted tongue, there is no great difference in the frequency of pyrexia; both may occur without it; the average temperature of the bare tongue is somewhat lower than that of the encrusted.

To sum up the circumstances of the red, smooth, dry tongue, they are usually more chronic than those of the encrusted. This must be so, since the one is often a later stage of the other. Exhaustion by discharge is frequent with the smooth tongue; or, if this be absent, there are generally especial circumstances of prostration or depression, which are often connected with the abdominal organs. This tongue implies failing nutrition. The clinical evidence accords with the indications of pathology in this respect. The saliva is, I believe, always deficient, though I think it is not usually so completely absent as with the encrusted tongue. It will presently appear that in certain circumstances want of saliva prevents the growth of epithelium; though this failure of growth may also have its connection with a more general impairment of the nutritive process. Increased moisture of the tongue is a sign of the best omen; this is usually followed by re-covering, and possibly by recovery.

The frequency of the red, smooth, or raw-beef tongue in connection with dysentery, especially where abscess of the liver is also present, and with other affections of the alimentary canal, cannot but attract especial notice, and has been attributed to reflex nervous influence. This cannot be absolutely negatived, yet we may hesitate to introduce such an explanation in the presence of other circumstances, which certainly go far to account for the state of tongue. The chief of these

are the prostration common in the disorder mentioned, presumably impairing the epithelial growth, together with the want of saliva, which contributes to the same deficiency, and the irritation of the unprotected surface by the contents of the mouth.

The prognosis with this tongue is bad: of the thirty cases in which it was recorded, sixteen ended fatally. The mind commonly remains clear, though the patient may be weak unto death.

10 and 11. BARE, SMOOTH, DRY, RED (MEMBRANE OR NONE EVIDENT.)

Pneumonia	5
Broncho-pneumonia (also after recovery)	1
Phthisis or general tuberculosis, or of peritoneum	4
Hepatic ascites and dry diet	2
Attack of gall-stones	1
Lardaceous disease, ascites, etc.	2
Dysentery and abscess of liver	2
Surfeit	1
Diabetes, advanced	5
Typhoid	2
Pyæmia	1
Lumbar abscess, discharging	1
Pus in urine (abdominal tumor)	1
Tubercular pyelitis, pus in urine	1
Empyema, discharging	1
(Total	30)

PARTICULARS RELATING TO THE ABOVE CASES.

Pyrexia (temp. 102° to 104°)	11
Hyperpyrexia (temp. over 104°)	2
No observations as to temperature	1
Average temperature in 29 cases	99.6°
Much prostration	21
Prostration not severe	2
Chiefly on liquid diet	6
Strictly limited to liquid diet	11
Dry diet	2
Saliva abnormally deficient	15

Died, 16; recovered, 6; relieved, 7; not relieved, 1.

CLASS 12.—CYANOSIS OR VENOUS CONGESTION OF THE TONGUE.

(*See Plate IV. No.* 12.)

This tongue is characterized mainly by a superabundance of recent or deep epithelium which occurs in circumstances

indicative of excessive production, not deficient removal. It especially belongs to heart-disease or cyanosis from some other cause. To the naked eye there is a bluish or purple color, and a smooth, wet, slippery surface like that of an eel, upon which the papillæ are almost indistinguishable, as if fused together or in some other way deprived of their separating intervals. Upon this substratum may be overlaid more or less of one of the grades of coat which have been described, to the partial obscuring of the deeper characters. Microscopically, the most noticeable peculiarities are superabundance of the recent epithelium, vascular injection, and hypernucleation. The deep epithelium is amassed thickly over the surface, sometimes rising to the tops of the papillæ, but leaving these exposed, sometimes overlaying them completely, so that the whole papillary structure is embedded. The Malpighian layer shares in the overgrowth. The horny epithelium is usually deficient, as also are superficial vegetations, so that the surface is often abnormally clean and smooth. The corium and deeper parts are enormously over-injected and over-nucleated, and the fibrous tissue of the corium is often coarse and hypertrophied. The appearances presented suggest the means by which they are produced. In the excess of deep epithelium and of nucleation we see evidence of increased growth, while the generally clean surface shows that wear is not wanting. The essential factor is a hypertrophic process connected with mechanical congestion.

The thick covering of the tongue with epithelium of the deep character is not confined to heart-disease or cyanosis. My classification was based on what can be surely recognized with the naked eye. Had microscopic examination been the criterion, a separate class must have been made, characterized by excess of epithelium of this sort. (See last figure on Plate IV.) But this peculiarity is not obvious. The horny or superficial epithelium when wet is white and conspicuous; the deep epithelium is neither. Its presence in excess may be inferred from the appearance of cyanosis, and guessed at from some degree of paleness and fullness. In a case of chronic albuminuria, where I had reason to think that this condition existed, though happily there was no opportunity of post-mortem verification, the tongue was remarkable chiefly for its pale color and enlarged papillæ. The pallor was such as to suggest

anæmia, a state which affected other parts as well. The tongue, was nearly clean, so that the papillæ were seen to advantage. Both filiform and fungiform were exaggerated in size, as could be seen with the naked eye and made more clear with a lens. The filiform looked fleshy and substantial. The absence of injection in the enlarged fungiform was in striking contrast with what is seen in other circumstances. Such a tongue may be thickly covered with superficial epithelium, by which the deeper characters may be hidden. Taking as the standard great excess of deep epithelium as displayed after death by the microscope, I find that of nineteen cases four were connected with cyanosis (cardiac or pulmonary), six with chronic albuminuria, two with acute tuberculosis, two with pyæmia, and one with each of the following—pneumonia, diphtheria, perityphlitis, lumbar abscess, and leucocythæmia. Thus we see this form of epithelial hypertrophy in circumstances of three kinds—venous congestion, albuminuria, and diseases attended with pyrexia. To take the last first, no details of temperature are needed when once the disorders have been named. The overgrowth was no doubt a direct result of the increase of body heat on principles which have been stated. No further reference need be made to the congestion of cyanosis. But I must halt for a moment upon the question of albuminuria. I have sections of eight tongues from cases of chronic renal disease, mostly the granular kidney; in six there was such accumulation as has been described; in two there was less, but enough to show the tendency. Why in these cases should the epithelium grow in such profusion? Not from pyrexia, for there usually was none; not from venous congestion, of which there was generally no sign. Was the hypertrophic process due to the increased arterial tension so constant in the circumstances? If this be so, we should find hypertrophy of epithelium elsewhere. The reason may admit of doubt; but I think none need apply to the observation that in chronic albuminuria certain parts of the lingual epithelium are remarkably increased.

Pityriasis Linguæ, or Annulus Migrans.

There is a curious affection which has been called ringworm of the tongue, pityriasis linguæ, lichenoid, and annulus

migrans. This is rare and incompletely understood. It must be considered as special to the tongue, for we know of nothing exactly like it elsewhere; but it is as yet uncertain whether it has its source in this organ or, as is perhaps more probable, is a manifestation of a disturbance arising somewhere else. It may not be properly included within my design, which is to deal with the symptomatic changes, but neither can it with confidence be excluded. I will therefore insert a word or two about it if only by way of note or parenthesis, more especially as I have had what I believe is a novel experience—the opportunity of post-mortem examination.

The tongue in this condition presents remarkable circles, or curves which may form parts of circles or be more or less irregular, with a general tendency to re-enter and enclose a space. Each circle or curve consists of a narrow white line of coating, while the central district is less coated or bare. The circles may be compared with tinea tonsurans, or "fairy rings" on turf, though with the incomplete curves the resemblance to either is less exact.

These patches, which are abruptly separated from the surrounding surface, which may be natural or nearly so, are often placed on the lateral portions of the tongue, though by no means always confined to them; more often than not they affect both sides, and that sometimes with exact symmetry, sometimes with an approach to it. Occasionally, as in the woodcuts, the eruption is displayed on the under as well as the upper surface. The patches begin in the centre and spread centrifugally, the white circumference marking the part last affected: the mode of progress cannot but suggest the advance of a fungus from a centre—a botanical hypothesis which, as will be seen, is not borne out by further examination. Scrapings of the surface during life display only what is found in most coated tongues—micrococci, filaments of oïdium and bacteria; but no special organism has come within my observation or been confidently described by others. Mr. Barker once found some cryptogamic structures resembling the trichophyton, but these were met with on one occasion only of many, and were thought to be accidental. The variety of organisms proper to the coat of the tongue must necessarily make it difficult to detect another; but not only has this not been done, but the general morbid anatomy of the change is

not consistent with a parasitic origin. The sections I have examined from the case I have alluded to show that there are alterations below the surface with which superficial parasites obviously have nothing to do.

The outer ring of coat presents long papillæ which are tipped with horny epithelium, and often surmounted with the vegetation common to the coating of the tongue. The central

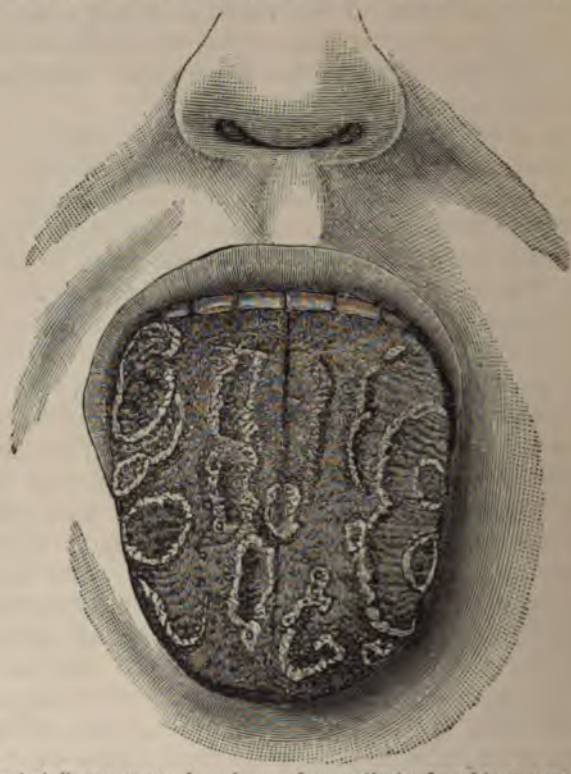

FIG. 5.—Pityriasis linguæ or annulus migrans, from patient referred to on page 650. Upper surface of the tongue.

bare part displays papillæ which have become denuded generally down to the Malpighian layer, so that all the superficial epithelium has gone. The Malpighian layer, or at least its deepest part, remains, though not unaltered, for the cells of which it consists are elongated as if overgrown. Below this layer in the corium are much vascular injection and abnormal profusion of leucocytes. These are collected in great abun-

dance along the margins of the injected vessels, in the superficial parts of the corium and within the central columns of the papillæ which are in continuation with this portion of the skin.

FIG. 6.—Lower aspect of the tongue from the same patient.

The extruded cells are in the greatest profusion in the central or enclosed part of the eruptive patch, where the papillæ have lost their covering and the surface of the tongue is bare. Out-

FIG. 7.—Section of tongue showing the eruption represented in preceding woodcuts. The white circumference shows an abundance of superficial epithelium. The whole section displays a profuse cellular formation in the corium, most abundantly in the bare central part of the affected patch. From a preparation made by Dr. Delépine.

side, in the surrounding ring, where the papillæ are fully, and even excessively clothed, the extrusion or cell-formation is less, but is present and is similarly disposed. The Malpighian layer shows general overgrowth, especially on the papillæ, where

the appearances suggest that this sprouting from below was the means of lifting and displacing the epithelium above, and so detaching the horny portion, the absence of which made the central region bare.

Thus the change appears to belong essentially to the corium, not to the exposed surface; to originate in the blood-vessels rather than in the epithelium, the changes in which are secondary to those beneath. The visible part of the disease appears to be one or more rapidly expanding foci of inflammation, or at least of injection and extrusion, which begin in the subepithelial part of the skin and carry superficial changes in their wake. The appearances when looked at minutely are more suggestive of some passing disturbance belonging to the blood-vessels than of a parasite, of which the nidus would presumably be in the epithelium. The bilateral symmetry, which is occasionally exact, is suggestive of a constitutional origin rather than a local one.

The eruption is extremely evanescent, changing from day to day. It affects the same person repeatedly. The woodcut represents an attack which was believed to be the eleventh. The clinical relations have as yet thrown but little light upon its nature. Of three cases I have seen, the subject of one, that from which the drawing was taken, was a woman, thirty five years of age, under the care of Dr. Whipham, who died of valvular disease of the heart of rheumatic origin. The subject of another was an anæmic little girl, four years old, who was liable to asthma. The third patient was a neurotic gentleman, approaching the age of sixty, who was the subject of arthritic pains, which were regarded as rheumatic, but which may have been gouty.

The pathological nature of the change shows that the blood-vessels are its immediate source, whatever may be behind to determine their action. Embolism at once suggests itself, as soon to be discarded. The lingual arteries arise from separate carotids, and these from different parts of the greater channels. It is improbable that the two linguals should often be similarly and at the same time penetrated by clots from a distance, and impossible that they should habitually be so. Putting embolism aside, we may next appeal to the bilateral symmetry in evidence that the change is not the result of a local irritant; thus a diseased tooth, or what not, could scarcely

be supposed to act so regularly and equally on both sides as must the cause of this eruption. Looking for general or remote causes, the circumstances are more suggestive of one acting through the blood than through the nerves. Blood disorders, the exanthemata, gout, rheumatism, and syphilis, continually affect both sides similarly; and though there are instances of central nervous disorders—locomotor ataxy for example—affecting the skin and tissues on both sides alike, yet many complaints of nervous origin, such as several forms of herpes, present themselves on one side only. It is clear that if this peculiar disease of the tongue originates in the nervous system it must be in some central part, but it seems more likely that it may have to do with the state of the blood or be akin to the disorders which are thought to have their chief concern with it. It is too soon to push this speculation into detail. Syphilis appears to be clearly excluded. Mr. Barker has referred to eleven cases of the kind—six in females, five in males, while of nine the subjects were under twenty years of age.[1] The absence of male preponderance and the incidence as regards age makes gout less probable than rheumatism. Of the three cases I have had under observation, one was associated with rheumatic disease of the heart, and another with a liability of which the nature as between gout and rheumatism was doubtful. Further experience is needed before more can be said.

I have now concluded the separate consideration of each variety of tongue. I shall proceed to make a few observations relating to some general conditions which belong to it or affect it, and while I do so, I must ask the College to tolerate a certain amount of repetition which it will not be possible to avoid.

Having regard to the importance of *dryness* as necessary to some of the most significant alterations of the tongue and its value *per se* as a clinical indication, I shall take this quality into consideration, abstracted, as far as may be, from others. Before doing so I shall venture to delay the College with a few observations upon the saliva.

On the Saliva.

I am satisfied that of all the immediate causes which make the tongue dry, or tend to do so, arrest in the secretion of saliva is the most important. In advanced conditions of dry-

[1] "System of Surgery," by Holmes and Hulke, 3rd edit., vol. ii. p. 560.

ness, not only is saliva obviously absent from the tongue and mouth, but the patient cannot spit. When the dryness is less complete he will sometimes produce with difficulty a little spittle, which is often tenacious or blood-stained. Frequently in the absence of saliva such patients will, on the request to produce it, hawk up a little mucus from the throat, which is clearly a different thing. I have made many experiments upon the salivary ducts, or rather, I should say, upon those of the parotid, which lend themselves most readily to the purpose. A tube passed into one of these channels in health could generally be made to drip saliva somewhat abundantly by the application of acetic acid to the tongue, or the secretion could be seen working its way out by the side of the tube. When the tongue was dry it was usually impossible to obtain any by such means. With advanced and complete dryness I have never been able to get a drop; with lesser degrees often a little. These observations show the dryness to be, as a rule, due to deficient secretion of saliva, not to its drying on the tongue, as has been supposed by some. This failure of secretion appears to be of much significance. It is not possible thus to gauge any other of the secretions which are concerned in the digestive process; but it is, at least, probable that the gastric and others may be similarly affected. It is a matter of immemorial experience that the dry tongue, more particularly that which is also encrusted, is incompatible with the digestion of solid food; the physician orders liquids and stimulants. This ancient practice has been arrived at not by reasoning but by a less unsafe though a longer road—experience. We may be sure that there is natural truth at its foundation, though we may not know what that truth is. Provisionally we may assume that a deficiency of saliva, if it depend on other than local causes, goes with a deficiency of other juices concerned in digestion. At the same time it would seem that other secretions, those, for example, of the kidneys, the intestines, and the skin—do not take part in any failure of secretion which may be supposed to exist. Putting aside cases where an excess of some secretion, as with diarrhœa or diabetes, has been contributory to the dryness, there are many of daily experience where together with a dry tongue the urine is fairly abundant, the bowels free, and the secretion of the skin not wanting or even excessive. If, therefore, it be presumed that

the failure of the salivary secretion is associated with a similar failure elsewhere, it is clear that the arrest is limited, not common to all the glands of the body.

I may add a few words upon the action of saliva upon the coat of the tongue; we have seen that, as a general rule, as the tongue becomes more and more coated, especially with acute disease, it gets drier and drier. This association of want of saliva with increasing coat suggests the question whether the secretion has any solvent action on the coat, so that this thickens from the absence of normal solution. This must be answered in the negative. I have taken coated tongues and kept similar pieces of them, one in saliva and one in water, for periods ranging up to sixty-eight hours. Then, making sections of each in the usual way, I have satisfied myself that there was no greater loss of epithelium in saliva than in water, and the same result has been given when the experiment was conducted at a temperature of 100° as at the ordinary temperature of a room. Whatever effect, therefore, is to be attributed to want of saliva, it is not by loss of any solvent action.

Other causes have been shown to conspire to make the tongue coated, but it would seem that want of saliva by itself will do this. A boy was brought under the care of my colleague, Mr. Rouse, with, as was believed, a fracture of the base of the skull. He had left facial paralysis, and an injury apparently of the chorda tympani on this side. The tongue was normally protruded, but the left half, sharply defined by the median line, was dry, and presented a milky appearance. The suppression of saliva was probably the cause of the coat, though other effects of nerve-injury could not in this case be absolutely excluded.[1] I will take a simpler case reported by Mr. St. Clair Buxton. A lady had mumps, after which her mouth became quite dry. The tongue, gums, cheeks, palate, and pharynx presented a "fearfully dried-up state." The tongue was thickly coated with a tough brown fur, which was horn-like and so hard that it sounded under a blow like the cover of a book. After ineffectual treatment by other means, the salivary ducts were probed, the glands galvanized, and the secretion restored. In the course of the following day, Mr. Buxton tells me, in a letter for which I have to thank him, the

[1] For this observation I have to thank Mr. Peel Davies, then house surgeon.

fur became soft and spongy, though it had previously been as hard and dry as wood; in a week all had cleared off, and in a fortnight or three weeks the tongue was perfectly normal. The fur stripped off in patches, leaving a rather raw surface underneath, which gradually assumed a natural appearance. It has been shown how closely the conditions of furring and incrustation are associated with deficiency of saliva, and inferred that these are largely the physical results of the ensuing dryness; the normally wet surface is soft and liable to wear; deprive it of moisture, and it becomes horny and resistant of it; besides which, with no saliva solid food is unacceptable. So that while induration is created attrition is reduced; and in general terms, and up to a certain point, it holds good that the less saliva the more coat.

But there is another relation between the secretion and the covering, and that of an opposite kind. An interesting case of lasting suppression of saliva was published by Mr. Rowlands, of Great Crosby [1] and I have to thank him for further particulars by letter. Ten years before the date of the report, a lady of the age of sixty underwent sudden and complete suppression of the saliva, after a nervous shock. Since this the mouth and tongue have been perfectly dry. The tongue now presents the appearance of a piece of dry raw beef; it is perfectly clean and intersected with superficial fissures; no papillæ are to be seen with the naked eye, though to be made out with a lens. Here is a typical red, dry, smooth tongue sequent, and probably consequent, upon simple deficiency of saliva. It is scarcely necessary to revert to the red smooth tongue of disease, of which deficiency of saliva has been shown to be a constant, if not a necessary, attendant. These experiences showing coating and denudation from the same cause are not so contrary as at first sight appears, for many smooth tongues were formerly rough. The difference is largely one of time: furring and incrustation are early results of want of saliva, denudation a late result. It has been shown that with the bare tongue of constitutional disease two agents are commonly present—want of saliva and failure of general nutrition: but that want of saliva by itself is able to cause bareness is shown by such cases as that reported by Mr. Rowlands. Of

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the *modus operandi* I was long in doubt. The epithelium is fed by the blood, not by the saliva. Why does it waste in the absence of the non-nutritive fluid? Conversation with my colleague, Mr. Bennett, has, I think, given the clue. The epithelium, though not nourished by the saliva, needs to be kept moist by it, otherwise it cannot assimilate what is provided by the blood. Be this as it may, a general law may be formulated: the tongue tends to become smooth whenever for a long time it is kept dry.

LECTURE III.

DRYNESS.

MR. PRESIDENT AND GENTLEMEN,—I now come to the consideration of dryness of the tongue and its causes. A glance at the annexed Table will suffice to show how largely this depends on constitutional and how little on local circumstances.

CONCOMITANTS OF DRYNESS OF THE TONGUE.

	Cases.
Tongue dry, irrespective of other qualities	113
Pyrexia (temperature 102° or over)	37
Temperature not above normal	39
Discharge by simple diarrhœa or dysentery	6
Discharge, diarrhœal or other, connected with lardaceous disease	2
Diarrhœa in connection with other forms of organic disease—phthisis, cirrhosis, etc.	9
Chyluria	1
Discharge by suppuration	8
Serous discharge by frequent tapping of pleura	1
Diabetes (including 3 of diabetic coma)	8
Dry diet	6
Coma or unconsciousness, with general openness of mouth	14
Openness of mouth from obstruction of nose, tonsillitis, or other causes unconnected with coma	6(?)
Great prostration or exhaustion	51
Cases ending fatally	56

The old physicians recognized dryness of the tongue as of evil omen. Hippocrates refers more than once to this as a bad sign. The great Willis speaks of the dry tongue in certain cases of fever, and refers it to constitutional causes, as expressed in the fanciful language of his time. "The nervous juice," he says, "is thoroughly roasted by a long concoction, and so becomes almost like glue, thick; wherefore not being able to be dispersed neither by spittle nor by insensible transpira-

tion, nor to be separated by the urinary passages, at length leisurely runs out by the passages of the spittle, and forthwith by reason of its thickness grows into that glueiness." I think most physicians at the present time regard the dry tongue as a constitutional symptom, and as a bad one, notwithstanding that there are some who have sought to refer it rather to local conditions than to the system at large. My cases, collected without special selection, show with the dry tongue, be its origin what it may, a startling mortality—one of almost exactly fifty per cent. Of 113 patients with dry tongues, fifty-six died.

I will now come to the causes in detail. The immediate causes are two: increased evaporation from the mouth; diminished secretion into it.

1. *Increased evaporation* may be due to exposure of the mouth by persistent openness; as when the nose is obstructed, and when coma exists; or to increased heat of body, and as a consequence of expired air. Further, there is a double relation between dryness and bareness: dryness has been seen to cause bareness; but must not bareness at least help to cause dryness? Epithelium is conservative of moisture, as may be witnessed in any dissecting-room; wherever the surface of the skin has been rubbed off the deeper part dries and cornifies. Thus, if by chance the upper epithelium of the tongue should be lost, the deeper investments must be more amenable to evaporation and desiccation.

2. *Diminished secretion* may be either of the salivary or the mucous glands; but the moisture of the tongue depends more upon the salivary secretion, which is abundant and watery, than upon the mucus, which is scanty and thick.

From the immediate conditions which determine dryness of the tongue I now approach the more complicated morbid circumstances which lie behind them. First, I will take conditions which entail persistent openness of the mouth, and so increased evaporation. Secondly, states of pyrexia, of which the bearing is more complex, but which involve increased evaporation from the tongue by reason of the heat of the expired air, and also general dehydration and consequent diminution of saliva from the generally increased evaporation which the heat of the whole body entails. Thirdly, profuse discharges which consist largely of water, by which the body

is dehydrated and the salivary glands in particular stinted of their proper material. Fourthly, conditions of prostration or exhaustion, which states are adequate in themselves to suppress the saliva and dry the tongue, though often they are assisted by other causes, more especially discharges and pyrexia.

Habitual Openness of the Mouth.—Much importance—I think I can show too much—has been attached to this either as occurring during sleep, or from obstruction of the nasal passages, or from coma. If the tongue be persistently and generally dry, I believe that other causes are always at work. I have known the tongue to remain perfectly moist while the nostrils were, and had been for some days, plugged for epistaxis. On the other hand, it is common to find a path of dryness down the centre of the tongue, corresponding to the exposure to breath in tonsillitis and other throat affections. With regard to the chronic enlarged tonsils of childhood, when the child sleeps with the mouth open, I am assured by my colleague Mr. Bennett, who has removed many of these, and always does it early in the morning, that on waking the tongue is usually dry, but afterwards soon becomes moist. Certainly such tongues are usually moist when they come under my notice. If the tongue is persistently dry from enlarged tonsils, I think the affection is usually inflammatory, so that causes other than exposure assist. In the earlier part of this inquiry it was my rule to examine into the course of the breath whenever I found the tongue to be dry; but the conviction was forced upon me that the state was generally due to other than mechanical causes, and I ceased to look for this one with the attention I had before given to it. My figures are therefore less reliable than the general impression which I record. In the great proportion of cases where the mouth is habitually open, it is either from coma or some illness which interferes with full and alert consciousness; the relaxed jaw and gaping mouth are but signs of ebbing vitality, of which the failure of secretion is also a part. I have sometimes ascertained by catheterization, what was obvious without, that the tongue in such cases is dry, not because normally abundant saliva has dried upon it, but because the secretion has been wanting. In cases where the tongue is dry and the mouth open, other circumstances generally concur in causing the condition: as in a case of tonsillitis with a temperature of 103°; one of acute

rheumatism with a temperature of 101.5°; one of suppression of urine with profuse diarrhœa; and I might add others. But I need not dwell further upon a cause of dryness which, though it has a place, has not an important one; nor does it obscure to any considerable extent the great constitutional significance of the sign in question.

Pyrexia.—To take this next, dryness of the tongue is so frequent with it that the two must be connected; at the same time, the occurrence of the dryness with little or no pyrexia shows that other causes may produce it. As the temperature of the body rises the moisture of the tongue diminishes; on the other hand, it by no means follows that if the tongue be dry the temperature is, or has recently been, raised. Many causes intervene which complicate the relation between heat of body and dryness of tongue. With typhoid or acute rheumatism the tongue is apt to be dry at a temperature under which, with pneumonia, it would probably not be so. Something is due to time; the more chronic disease is the more drying. A temperature of 102° has little effect in drying the tongue. Even much higher temperatures are reached without the tongue becoming dry, though its moisture is lessened. I have noted many instances of acute disease where the plastered tongue has retained enough moisture to forbid its being called dry under temperatures of from 103° to 104°. It would appear that over 104° dryness is general; over 105° nearly constant. The extent to which other causes intervene is shown by the annexed statement, showing the maximum temperatures in 103 cases of dry tongue.

Temperatures subnormal in	. . . . .	16
" ranging up to normal in	. . . .	19
" " " 100° in	. . . . .	16
" " " 102° "	. . . . .	28
" " " 103° "	. . . . .	15
" " " 104° "	. . . . .	5
" " " 105° "	. . . . .	3
" " over 105° "	. . . . .	1
(Total	. . . . 103)	

In a third of the number the temperature was not over normal; in half it was not over 100°. There is no variety of dry tongue which gives so high a mean temperature as the moist plastered tongue of acute disease. It is clear, then, that

though pyrexia is a drying agent, yet there are others which are important.

General Dehydration.—Next, as a cause of dryness of the tongue, I come to general dehydration of the body, whether by deprivation of drink or excessive aqueous discharges. Apart from complications, we presumably have to do not with increased evaporation from the mouth, but with diminution of the saliva, often as a simple and direct result.

First, as to deprivation of water. I have never seen this carried to extremity, nor can I adopt as my own the experience of "The Ancient Mariner:"

> "And every tongue through utter drought
> Was withered at the root;
> We could not speak no more than if
> We had been choked with soot."

But of lesser degrees of deprivation, mostly in the treatment of aneurism and some forms of dropsy, I have recorded eleven instances, in six of which the tongue became dry. I need say no more of these cases now than that they show the results of dehydration in its simplest form; diminution of saliva and dryness of the tongue, with sometimes furring, sometimes more or less denudation.

As a mode of dehydration less simple but more definitely morbid I may next refer to diarrhœa. Of the 113 cases of dryness this flux, simple or complicated, was present in seventeen. I here exclude typhoid. Six were cases of simple or dysenteric diarrhœa; the rest were associated with phthisis, cirrhosis of the liver, lardaceous or other organic diseases, which it would serve no purpose to recapitulate. In six the temperature was 102° or over; in five not above normal. In the larger number the complications were such that it was impossible to assign the state of tongue to the diarrhœa only; but there were three which served to exemplify the dryness as a direct result of diarrhœa, without either organic disease or marked pyrexia; in one of these the temperature was subnormal. More often when this appearance presents itself under the flux some other condition is present, often organic disease, the fatal end of which is not far off.

The maintenance of the moisture of the tongue under diarrhœa, profuse even to death, is a matter of common experience

in Asiatic cholera. The tongue here remains moist, creamy, and noticeably cold during the whole of the purging or algide stage. When the discharges have ceased and reaction and fever set in, then as the pulse rises the tongue[1] begins to dry and becomes quite dry and brown, as often in typhoid, the edges and the tip being red. Probably the persistent moisture of the algide tongue is in some measure due to the watery vomit which so abundantly flows over it; but something must also be attributed to its low temperature. It is a matter of experience that a tongue which is in itself dry is not easily kept otherwise by external wetting. The patient may drink often to this end, but only with a superficial and evanescent effect.

Excessive discharges of urine may be next considered. Diabetes mellitus is a cause of extreme dryness of the tongue; of the 113 cases where this state of tongue was recorded, eight were of this disease. When the tongue has been dry under a mixed diet it will often become moist under a restricted one. By restriction both the sugar and the water of the urine are lessened; the formation of sugar is checked, and with this the discharge of water. Whether the presence of sugar in the blood or the discharge of water takes the chief part in drying the tongue may be ascertained by a comparison with diabetes insipidus, where the loss of water occurs without the production of sugar. I have notes of eight cases of this kind, in most of which the discharge of water was greater than with diabetes mellitus. The tongue was recorded as moist, generally slightly coated, in six; in one as "cleaning;" in another as "dryish, pale, and flabby." The desiccation was therefore decidedly less with diabetes insipidus than with diabetes mellitus, and in the latter disorder must therefore be attributed to some other cause than the simple loss of water by the kidneys. I may mention a case which bears on this point. A woman had profuse diabetes insipidus, passing on an average a pint of urine an hour for the twenty-four hours, and drinking to almost exactly the same amount. The tongue remained moist, slightly coated, scarcely unnatural, except that it displayed a syphilitic scar. Three years afterwards I saw the

[1] In this description of the tongue in cholera I have been enabled to supplement my own recollections by the more extensive experience of Surgeon-General Cornish.

patient again, having lost sight of her in the interval. A very unusual change had taken place; the diabetes had altered from *insipidus* to *mellitus;* the urine was now loaded with sugar and in exactly half the quantity, the amount of drink having fallen in the same proportion. The tongue was now dryish, reddish, and a little brown. The temperature of the body was subnormal. The patient was on the verge of diabetic coma, in which she shortly died. The access of dryness of the tongue with glycosuria, though the diuresis was diminished and the temperature not increased, was instructive. The osmotic action of the sugar in the blood is probably the chief cause of the dryness of the tongue in the circumstances, as it has been shown to be of the dehydration of the lens and consequent cataract. The proximate cause of the dryness of the tongue is absence of saliva, as is evident by the state of the mouth and of the parotid.

Prostration.—In speaking of the several varieties of dry tongue, I have shown how much fatal disease they present—altogether about fifty per cent. A dry tongue, more than any other, foretells the ending of mortality. The kinds of disease which it accompanies are chronic more than acute; if febrile, usually continued. Putting aside designed restriction in drink, and also diabetes, where the dryness is due to special circumstances, one is at once struck with the gravity of the cases and the large proportion of these which end fatally. The conditions are most various; it is not easy to see what they have in common, except it be something which may be indicated by such terms as *prostration* and *exhaustion.* Exhaustion by suppuration of many sources, advanced phthisis and tuberculosis of other kinds, are frequent when the tongue is dry and smooth. When it is dry and rough, the tables show concluding brain disease, concluding cirrhosis, advanced cancer, advanced pyæmia, and severe pneumonia. Of twelve cases of pneumonia in which the tongue was dry seven ended fatally; of twenty-seven in which the tongue was moist only five ended fatally. A dry tongue in rheumatic fever is commonly recognized as an unfavorable omen, though not necessarily a fatal one. In typhoid the converse may be stated—a persistently moist tongue is indicative of a mild attack.

I have used the term *prostration* as a somewhat inclusive one—one which can be better understood than defined,—repre-

senting great failure of strength and nutrition, however brought about. With the 113 cases of dry tongue, this condition was noted in fifty-one; in 222 cases where the tongue was moist, it was noted in but twenty-four. We cannot but conclude that prostration or failure of vital force is the most important factor of the dry tongue; pyrexia takes the second place. Clinical experience warrants the assertion that, though dryness of the tongue may occur without great prostration, great prostration is never long continued without dryness of the tongue. Great prostration occurs with little alteration of the tongue in connection with abdominal collapse, as in acute obstruction and perforation, but the condition has not been of long continuance.

Besides prostration—or, be it rather said, together with it —coma presents itself in connection with lingual dryness; this is not wholly due in the circumstances to the open mouth, as I have already shown, but to deficient secretion of saliva. It is to this that the dryness of the tongue is usually due, and it is because the dryness is an index of this deficiency that it has the clinical importance which, I think I have shown, must be attributed to it.

On the Influence of Food upon the Tongue.

In considering the causes of the several states of tongue, more particularly of dryness, I have said much which I need not repeat as to various influences which bear upon it. But there are still one or two which demand separate consideration, however brief.

First comes the question of food. The act of eating undoubtedly has an effect in cleaning the tongue, which is mechanical; and coating has been thought to depend, more largely than upon any other circumstance, upon the absence of the attrition which this process entails. It has been shown that the tongue is commonly more coated before food than after, that it is apt to be coated on the side of a tender tooth where mastication is limited, while it remains clean on the sound side where it is not; and much of the effect of acute disease in coating the tongue has been ascribed to the attendant loss of appetite and restriction of diet. But I have already shown that coating is a matter not only of want of wear, but

in part of overgrowth; and that other causes (notably pyrexia) are directly concerned. As regards the influence of food, I have sought instruction in cases where there was absence or limitation of it, apart from pyrexia or other causes which act upon the tongue. I have watched the state of this organ in many cases of stricture of the œsophagus where solid food has been entirely disused, and have before me the notes of five such, and I might add as a sixth a case in which the patient refused food in consequence of cancer of the larynx. The back of the tongue here was covered with long, shaggy, fur-like, coarse hair. (See Pate II. 7D.) There was another instance in which the tongue was furred, but it did not become so, notwithstanding long total absence of solids, until it became dry under extreme prostration and absolute pulselessness. Among the other cases there was no instance (though, in some, solids by the mouth were impracticable and feeding conducted chiefly by the bowel) in which the higher degrees of coat existed. In two the tongue was coated, but not plastered; in one it was partly stippled or dotted, being coated only in the back and central parts; in one it was dotted only. In the last case, slight as the covering was, the difficulty of taking food was such as to call for gastrostomy. This case declared in a manner to which accident gave effect how slight is the coating produced by absence of food, as compared with that due to acute febrile disease; for it chanced that I had at the same time in near proximity some typical cases of the plastered tongue of pneumonia and pyæmia. The difference was graphically displayed; among other points the general spread of the acute coat over the dorsum was contrasted with the tendency of the other to collect at the back and in the median line, leaving much of the tongue nearly free.

Cases of restriction to liquid diet, not as a mechanical but a physiological necessity, are seen daily. The physician knows that no other will "agree with" the patient; he is guided chiefly by the presence of pyrexia and the state of the tongue: the more coated the tongue, the more liquid the diet; if the tongue be dry, the diet is wholly liquid and alcohol part of it. Here the tongue determines the diet, not the diet the tongue; but not without instruction is the issue. As the acute disease abates, the tongue cleans notwithstanding the limitation: as it cleans and because it cleans, solids are added and may help

the process; but the cleaning comes first. In my table of normal tongues are seven which were so under a strictly liquid diet; on every ground, therefore, it appears clear that, though some influence must be ascribed to food and mastication in cleaning the tongue, yet these are of secondary importance.

I need not revert to the effects of dry diet, which have been discussed in relation to dryness of the tongue; these are briefly want of saliva; and in some cases furring, in others denudation.

On Conditions of the Alimentary Canal in relation to the Tongue.

It is a common belief that the tongue is directly indicative of many disturbances of the stomach, bowels, and organs directly connected with digestion; some appear even to be possessed with the fancy that the tongue is but an exposed sample of the alimentary canal, and declares by its changes the existence of similar changes in the hidden parts. With those who do not go thus far the white tongue is taken as a sign of constipation, or that the stomach or the liver is out of order, and that alteratives, especially of the mercurial kind, are needed. It is not easy to disentangle the complications which involve this subject; in the endeavor to do so I must appeal to a wider experience than I have been able to tabulate.

First as to the stomach. I have examined this organ after death with the naked eye and with the microscope in many cases where the tongue has been thickly coated or furred. Examination of the stomach is unsatisfactory partly from the post-mortem influence of its contents; but it may be safely said that this organ presents no changes which are obviously analogous to those of the tongue, and the same statement may be extended to the rest of the alimentary canal.

I have not been able to discern any state of tongue especially connected with dyspepsia or ulcer of the stomach. When dyspepsia is associated with stomatitis the tongue is sometimes thickly coated, probably as a local result. In simple dyspepsia and ulcer, the lower degrees of coating are usually present, possibly in connection with the loss of appetite and the limitation of food. In one case of ulcer I noted the tongue as clean but flabby.

Next as to the bowels. Some forms of constipation or diseases associated with it are undoubtedly connected with changes in the tongue; but that the arrest is not necessarily connected with any such changes is evident. I have seen the tongue perfectly clean and normal after three weeks of nearly total constipation in a hysterical woman, and equally so after twenty-eight days of nearly total constipation in a woman who was next day made the subject of colotomy for stricture of the sigmoid flexure; and it would not be difficult to cite other cases where the tongue has remained natural under long constipation, either functional or connected with chronic obstruction. On the other hand, where the obstruction is acute, the tongue early becomes stippled or coated and dry. I think the difference between the tongue of acute and of chronic obstruction, and between one case of chronic obstruction and another, is in the presence or absence of constitutional disturbance. The early dryness of acute obstruction is not, as a rule, associated with pyrexia, but depends on salivary deficiency associated with the constitutional state. The dryness determines the state of tongue. Unless there be constitutional disturbance, which with simple constipation or chronic obstruction there often is not, the tongue may remain natural. I have more than once noticed an old block in the rectum to be attended with thick coating, which has disappeared or lessened on the removal of the accumulation. It may be inferred that in such cases there is general disturbance and probably pyrexia connected with morbid absorption, as the fetor of the accumulation cannot fail to suggest.

Passing from constipation to the opposite condition, diarrhœa is early and powerfully productive of lingual changes. The tabulated cases speak for themselves, and I might largely add to the evidence they present. Looking through my notes not included in the tables, I find the tongue of diarrhœa described as "thickly coated and dry," "thickly coated, mouth dry, saliva scanty," "foul and coated," "brown, dry and furred," "coated with brownish fur," "very dry, brown in centre, coated," "dry, brown, and cracked." There is scarcely any condition in which the tongue becomes more rapidly dry, coated, furred, and encrusted than severe diarrhœa. The absence of saliva is self-evident; direct dehydration helps to cause this and pyrexia, which is often present, helps the desiccation.

I have already referred to the red and bare tongue which is sometimes associated with dysentery, together with abscess of the liver. Here we have fever of the hectic type together with the purging.

Opium.

It is no part of my design to describe the action of drugs upon the tongue, but I will nevertheless insert a word as to opium. It is well known that opium makes the mouth dry. I have frequently given it experimentally to persons whose tongues have been clean, or nearly so, and moist. The effect has always been to coat the tongue, impair the appetite, and diminish the saliva. In one case the temperature rose from normal to 100°. Some of the coating may be due to the loss of appetite and of friction, but the diminution of saliva must also have some importance attached to it, and falls in with what has been already said with regard to the relation of this secretion to the coat.

On the Influence of the Nervous System upon the Tongue.

This is not to be ignored, though I think more has been attributed to it than can be demonstrated. The late Mr. Hilton showed that coating of the tongue was often confined to the side of painful teeth, and referred this to reflex nervous irritation. I have already adverted to such cases, in the view that the effect was due to want of wear on the tender side. It is a matter of common observation that in hemiplegia the tongue is not unilaterally affected as if from the local change, but bilaterally as if from the constitutional results of it. On the other hand, I have already cited an instance in which there was reason to believe that unilateral coating and dryness of the tongue depended on an injury to the chorda tympani on the affected side. Here we have the intervention of saliva, and may with probability ascribe the coating to the want of this secretion rather than to the direct effect of nervous lesion. It has already been shown how largely deficiency of saliva affects the tongue, and it is a matter of ancient as well as modern experience that this secretion is much under the influence of the nervous system. I do not now refer to physiological experiments, but to conditions of wider range. The

mind affects the saliva, and may, or even must, by its means, or by means of its absence, affect the tongue. The dryness of agitation is well known; the tongue "cleaves to the roof of the mouth." The Eastern ordeal by rice, which can be swallowed by the innocent, not by the guilty, will occur to every one. I have often noticed the tongue to be white and sodden-looking after an exacerbation of nervousness, associated in particular with phosphatic urine, and thought the first change to be a want of saliva, the coating secondary. I was told by Mr. Charles Hawkins that a certain practitioner, who saw many men of business after their business hours, could always tell how things were in the City by the tongues of his patients.

The Tongue in relation to Individual Diseases.

I need not here discuss in relation to the tongue the general conditions of dehydration, pyrexia, and prostration, since to do so would be but to repeat what I have said in connection with coating and dryness. Neither need I delay, or only for a moment, to point out that if one were to seek to connect the states of the tongue as a general rule with individual diseases, so various, and apparently so contradictory, the results would be that nothing but confusion could result. What can be said towards this end may be briefly put. There is a tongue of heart disease of which the cyanotic character is evident to the naked eye, and a somewhat similar condition which is apt to accompany chronic albuminuria, which needs the microscope for its detection. The tongue of diabetes mellitus has a special tendency to dryness, as has been sufficiently explained; but this does not prevent its being sometimes normally moist or even normal in all respects. Among the febrile diseases it would commonly be said that the tongue of scarlatina and that of typhoid are characteristic. The well-known strawberry tongue of scarlatina approaches nearly to the pathognomonic, for the eruption helps to make it so; but even here other febrile disorders, like pneumonia, in which the face and mouth are apt to be injected, produce an excellent counterfeit, besides which the strawberry character in scarlatina is soon replaced by other phases of coating and by denudation. The tongue of typhoid presents many varieties, according to the stage of the disease and other circumstances. In eighteen cases it was

stippled and coated, coated, plastered, strawberry, furred, encrusted, and denuded. Only two presented the dry furred, or encrusted, state, which is commonly regarded as typical. The dry bare condition was not absent. The tongue of pyæmia more often shows the ideal typhoid state than does that of typhoid itself. I say nothing of the tongue of typhus, of which of late years I have seen but little. In the only case I have recently seen it was dry and black, as it is known often to be. Lobar pneumonia presents a range of tongue which runs through the whole gamut, the plastered type preponderating, as it does with typhoid and most other acute febrile states. In bronchitis the lower degrees of coating are generally presented; but if the disease be considerably febrile the tongue is apt to be plastered, which may be accepted as a sign of severity. With regard to acute rheumatism, the variety is considerable, the lower degrees of coating being more often seen than with diseases which present a higher temperature and more depression. Nevertheless, the dry furred and encrusted tongues, with their significations, are not absent from the series. It would serve no purpose, and would involve repetition, were I to dwell further upon the relations of the tongue to individual diseases; the tables speak in this sense, though the experience therein recorded is but a fragment small indeed compared to what must be in the minds of many who honor me with their presence to-day. The table now displayed (see pages 670, 671) is a compendium or consolidation of those already adduced separately in connection with each variety of tongue, and presents the cases which have been systematically tabulated. The kinds of tongue which were observed with each disease or state may be learned by reading horizontally; the several disorders and conditions which were recognized with each kind of tongue may be learned by reading vertically.

I have not dealt with local affections of the tongue, nor have I had much experience of them. That local irritation increases the coat may readily be believed. The coated or thickly stippled tongue of the smoker is well known; this may even assume—though, I trust, but rarely—the startling form of leucoplakia—an exaggeration, I presume, of the epithelial growth, though here I speak without post-mortem observation. I present a drawing from a patient with regard to whom I

TABLE SHOWING VARIETIES OF TONGUE AND DISEASE IN 366 PATIENTS.

	Natural	Stippled, Moist	Stippled, Dry	Stippled + Coated, Moist	Coated, White, Moist	Coated, White, Dry	Strawberry	White, Plastered	Furred, Moist	Furred, Dry	Encrusted, Dry	Becoming Bare	Denuded, Red, Dry	Cyanosal	Number of cases of each disease and condition
Cerebral hæmorrhage	..	..	..	1	1	..	..	..	..	..	..	..	..	..	2
" embolism	..	..	..	1	..	..	..	..	..	1	..	..	..	..	2
" disease with coma	..	..	..	..	..	..	..	..	..	2	..	..	..	..	2
" disease with hemiplegia or other paralysis	..	2	..	1	2	1	..	..	..	..	..	..	..	..	6
" giddiness, insanity, or tumor	..	..	..	1	..	..	..	1	..	..	..	1	..	..	3
Bulbar paralysis	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1
Sunstroke	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1
Acute myelitis	..	..	..	..	..	1	..	..	..	..	..	..	..	..	1
Tetanus	1	..	..	..	..	..	..	..	..	..	..	1	..	..	2
Neuralgia	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1
Chorea	2	1	..	1	..	..	..	..	..	1	..	..	..	..	5
Hysteria	..	2	..	2	..	..	..	..	..	..	..	..	..	..	4
Valvular disease of the heart	1	7	..	5	..	2	..	..	..	1	..	1	..	3	20
Intrathoracic aneurism	1	..	..	..	1	..	..	..	..	3	..	..	..	..	5
Pericarditis	..	1	..	1	..	..	..	..	..	..	1	..	..	..	3
Pneumonia and pleuro-pneumonia	2	4	..	2	6	2	2	8	2	..	2	4	5	..	39
Broncho-pneumonia	..	2	..	..	..	..	..	..	..	1	..	..	1	..	4
Pleurisy with effusion and hydrothorax	1	1	1	..	2	..	..	..	..	1	..	..	..	..	6
Empyema and pyo-pneumothorax	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1
Laryngitis	..	1	..	1	1	..	..	..	..	..	..	..	..	..	3
Bronchitis	..	2	..	1	2	..	..	3	..	..	..	..	..	2	10
Asthma	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1
Phthisis, including hæmoptysis and tuberculosis	1	1	..	3	3	1	..	1	..	..	1	..	4	..	15
Whooping cough	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1
Acute obstruction of bowel	..	1	1	..	1	1	..	1	..	..	..	..	..	..	5
Chronic " "	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1
Fæcal accumulation	..	1	..	..	..	..	..	..	..	..	1	..	..	..	2
Constipation, chronic	..	..	..	2	..	..	..	..	1	..	..	..	..	..	3
Colic (lead, &c.)	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1
Peritonitis or perityphlitis	..	..	1	..	1	..	1	1	1[1]	1	..	1[1]	..	..	7
Gastric ulcer, simple	..	1	..	1	1	..	..	..	..	..	..	..	..	..	3
Repletion, surfeit	..	..	..	..	1	1	..	..	..	..	..	..	1	..	3
Recent alcoholism	..	2	..	1	..	..	..	..	..	..	1	..	..	..	4
Dyspepsia, or with stomatitis	..	..	..	..	1	2	..	1	..	..	..	..	..	..	4
Sympathetic vomiting, uterine	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1
Simple or choleraic diarrhœa	..	..	..	1	2	..	..	..	..	2	1	..	..	..	6
Dysentery	..	1	..	..	..	..	..	2	..	..	..	..	..	..	3
Dysentery, with abscess of liver	..	..	..	..	..	..	..	..	..	..	..	1	2	..	3
Starvation	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1

Stricture of œsophagus, limiting food	..	..	..	1	2	..	..	..	..	1	..	..	..	..	4
Solids withheld	..	1	2	8	15	3	2	8	1	5	4	2	6	3	60
Limitation of drink for special reasons	3	3	1	1	..	..	..	..	..	3	..	..	2	..	13
Tonsillitis	..	1	..	..	..	1	..	..	..	..	..	..	..	..	2
Nostrils plugged, for epistaxis	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1
Cirrhosis of liver	..	1	..	1	..	..	..	..	.	2	..	1	2	..	7
Enlarged liver, nature uncertain	..	..	..	..	..	..	..	1	..	1	..	..	..	..	2
Obstructive jaundice, gall-stones, &c.	..	2	..	..	..	..	..	..	..	..	1	..	1	..	4
Ascites or anasarca, cause uncertain	1	..	..	2	..	..	..	..	..	..	..	..	..	..	3
Chronic albuminuria	2	5	1	6	1	1	..	..	..	..	..	..	..	..	16
Lardaceous disease	..	..	..	..	..	..	..	..	..	..	..	..	2	..	2
Uræmia	..	..	..	..	2	..	..	..	1	..	2	..	..	..	5
Diabetes mellitus	..	2	..	..	..	2	..	..	..	..	1	..	5	..	10
" insipidus	1	..	..	..	..	..	..	..	..	..	..	..	..	..	1
Hæmoglobinuria	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1
Chyluria	..	..	..	..	..	..	..	..	..	1	..	..	..	..	1
Acute rheumatism	..	5	..	7	1	5	..	3	..	1	1	..	..	..	23
Subacute, chronic rheumatism, and sciatica	..	2	..	4	4	..	..	..	..	1	..	..	..	..	11
Acute gout	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1
Chronic gout	..	3	..	3	..	..	..	..	..	..	..	..	..	..	6
Syphilis, congenital and tertiary	1	..	..	1	..	..	..	..	..	..	..	..	..	..	2
Lymphadenoma	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1
Addison's disease	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1
Internal cancer	..	..	..	..	1	..	..	..	..	..	2	1	..	..	4
Typhus	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1
Typhoid	.	..	..	3	4	..	1	5	..	1	1	1	2	..	18
Febricula, febrile attack, uncertain	..	..	..	..	1	..	..	1	..	1	..	..	..	..	3
Scarlatina	..	..	..	..	..	..	3	..	..	..	..	..	..	..	3
Mumps	..	..	..	..	..	..	..	1	..	..	..	..	..	..	1
Erysipelas	..	..	..	..	1	..	..	..	..	..	1	..	..	..	2
Measles	..	..	..	..	..	..	..	1	..	..	..	..	..	..	1
Pyæmia or septicæmia	..	..	..	..	..	..	..	2	..	..	3	1	1	..	7
Ague	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1
Eczema	..	..	..	2	..	..	..	..	..	..	..	..	..	..	2
Pemphigus	..	..	..	1	..	..	..	..	..	..	..	..	..	..	1
Erythema nodosum	..	..	..	1	1	..	..	..	..	..	..	..	..	..	2
Myxædema	..	..	..	..	..	..	..	..	..	..	..	..	..	1	1
Idiopathic anæmia	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1
Rapid emaciation, cause uncertain	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1
Anæmia, chlorotic, &c.	1	4	..	4	..	..	..	..	..	..	..	..	..	..	9
Pyrexia	2	3	..	6	9	6	4	15	1	3	8	5	11	..	73
Hyperpyrexia	..	..	..	..	..	..	..	3	..	..	1	1	2	..	7
Exhaustion from suppurative discharge	..	..	..	..	..	..	..	..	..	..	..	..	4	..	4
Suppuration without discharge	..	..	..	1	..	..	..	..	..	1	..	..	..	..	2
Great prostration from various causes	..	1	2	5	11	3	..	5	2	6	12	7	21	3	78
Convalescent	6	..	..	2	2	..	..	..	..	1	..	.	..	..	11
Total number of cases	24	62	4	69	48	20	7	32	5	24	22	13	30	6	366
Deaths	2	15	1	7	11	6	0	10	3	9	14	10	16	4	108

[1] Also perforation.

had the advantage of the opinion of Mr. Jonathan Hutchinson. I will dwell no further on this part of the subject, save to repeat what I hope has been made evident, that general influences tell more widely upon the tongue than local ones.

Conclusions.

It only remains that I should sum up briefly the conclusions which have been arrived at.

The tongue is an index of constitutional states, seldom of individual diseases. An ancient theologian described the face of a wicked man as a map of the empire of sin. It has been fancied that the tongue presents a map of the empire of disease; and a writer, though one of no great note, has gone so far as to divide the lingual surface into a number of rectangular regions as numerous as the United States of America, which he places under the rule of separate organs; the larynx, the bronchi, the lungs, the pleuræ, the large intestine, the small intestine, the kidneys, and the brain each possessing a distinct territory. The heart, says this writer very wisely, has a common control over all. But in truth the tongue has no such local signification; it seldom points to solitary organs or isolated disorders, but is rather a gauge of the effects of disease upon the system than an indication as to the locality of it. It is often a guide in treatment, so far as treatment is general, not local; and it is an important help in prognosis. It may, indeed, be doubted whether any means of observation open to the physician, including the pulse and the thermometer, give him more insight into constitutional states than he can derive from the tongue. Clinically it always speaks the truth, and in a language which is not foreign to the experienced physician. And how much truth, or rather how many truths, are to be read on how small a page! Conditions of fever and of feeding; states of the nervous system; the maintenance or abeyance of vital secretions; failure of vitality, though we may not be able to find out why; in one case that the disease is getting the better of the patient; in another that the patient is getting the better of the disease;—all these are discernible to the educated eye. The clinical value of the tongue largely depends on the number of interests it represents; these are more or less mingled in its indications, and

the impression they convey is a combined one; but it is none the less valuable because comprehensive; it gives to a glance what otherwise could be learned only by detailed inquiry.

The two factors which stand before all others in the making of the medical indications which the tongue presents, by which I imply those changes which are connected with remote or general states, not local disorders, are the heat of the body and the secretion of saliva.

It has been shown that the white coat of the tongue essentially consists of horny epithelium, and that the various grades of coating are mainly due to its increase. I have not dwelt upon the parasites which are apt to gather upon the coat; these are only of secondary interest; they do not determine the character of the coat or of the tongue, and they have been subjected to an exhaustive examination by Mr. Butlin, with results in negation of their practical importance. It has been shown that the several degrees of coat are mainly due to overgrowth of epithelium, though in a smaller measure to its want of removal, and that there is a remarkable correspondence between the heat of the body and the coating of the tongue. If the tongue be coated, the indication is usually of febrile disturbance without especial reference to the stomach or liver, and points more to the general system than the alimentary. Though something is to be ascribed to disuse, yet I think I have shown that too much has been; and that more than one modern observer, like an uncharitable pedagogue, has attributed to idleness what illness might truthfully explain.

Superadded to the *forcing process*, if I may so speak, of fever, we have step by step other changes, dryness, furring and incrustation, which are essentially connected with want of saliva. I have endeavored to show that this diminution or arrest is declared by the state of the tongue almost as certainly as by observations on the ducts, and is the chief cause of the furring and incrustation which accompany it. It is true that in the crust are parasites, but these are secondary; the primary fact is the want of saliva, a clinical indication always of importance, though the process by which it is brought about is not always the same. I have shown the effect of dehydration in diabetes and by diarrhœa, but it has, I think, been made clear that the most frequent and important concomitant of the dryness is a certain failure of bodily force

and function which I have not assumed to describe with physiological exactness, but have expressed by such terms as weakness, prostration, and exhaustion. I do not ignore the effects of deprivation of water, of alcohol, and of opium; but, nevertheless, the relation to which I have drawn attention, so far as it concerns such dryness as to cause incrustation, has a general hold. It is difficult not to infer that with the salivary are other glandular failures, more especially such as concern the digestive system. Good digestion waits on appetite. Putting aside diabetes, where there are special circumstances, it may be said with general truth that with the dry encrusted tongue appetite is *nil*, and solid food impossible, not merely from the local difficulty caused by the dryness of the mouth, but from inability more profoundly seated. It may be inferred without rashness that the loss of power to take food is connected with a loss of power to assimilate it; and if the digestive function is in abeyance it is not likely that the digestive fluids are abundant. Hence it presents itself as what may be called a working probability that a want of the more vital juices concerned in nutrition may be indicated by the want of saliva which is sometimes so conspicuously displayed. Physicians acknowledge in their practice some such guidance; the dry and encrusted tongue is seldom disregarded as a call for animal liquids, which require little digestion, and alcohol, which requires none. To translate theory into practice is not only dangerous because the theory may be wrong, but the means may be ill adapted though the theory be sound. I have often taken the dry tongue as an indication for peptonized food, and thought it beneficial, but have not as yet had enough experience to speak confidently.

Proceeding from the varieties of clothing which dryness produces, we come to the opposite, but sometimes succeeding, condition—that of nakedness. This is often connected, like the previous, as has been shown, with want of saliva, of which it is usually a later concomitant. It may be simply due to this cause, but other circumstances are so often present that it is difficult not to assign to them some share in the loss of integument, and attribute this, in part at least, to the failure of nutrition which belongs to hectic fever and suppurative waste. When the tongue becomes dry and bare, it is ill with the patient. He is not sure to die, but likely to. If, as has

been said, the tongues of dying men enforce attention, it must be often directed to this. The indication of the red, smooth tongue is for what failing nutrition calls for—tonics, stimulants, and food, probably liquid, but nourishing. The failing pulse does not more surely tell of asthenic tendencies than, as a rule, does the red, dry, and polished tongue.

The tongue, indeed, has a whole book of prognostics written upon its surface. A glance may suffice to show whether it is on the road towards health or from it, and thus tell us at once what could otherwise be ascertained, and then less surely, only by a historical research. When the tongue is approaching the condition of health, so, as a rule, is the patient, as is seen whenever the red, dry, and bare tongue acquires moisture and clothing. In fevers the gradual recovery by the tongue of moisture and the exposure of a normal surface at the tip and edges as the coat recedes are favorable indications of the greatest significance. There is no better sign in diabetes than the resumption of the natural moisture by a tongue which has been dry. Something may be judged by the way an encrusted tongue cleans; if gradually and from the edges, well; less so when it scales, especially when the surface exposed is red and dry. One glance at a coated or plastered tongue may give an assurance, which perhaps could not be otherwise obtained, that the disease is on the wane. If the thick coat in the centre steeply shelves towards the sides and front, revealing a normal, moist, not over-injected surface, the tongue is in process of cleaning; the natural friction is overcoming the coating process, and tongue and patient are on the mend. A tongue acquires coat more evenly and generally than it parts with it; we can thus tell whether the coating is on the advance or decline, and apply this rule to the disease.

There remains to me only the pleasant duty of mentioning those to whom I have not yet referred to whom I have been indebted. No one who has worked at the tongue can fail to have profited by the labors of Mr. Jonathan Hutchinson, to whom, indeed, I have been under special obligation, though as surgeon and physician our points of view have not been the same. I must next record my debt to Mr. Sweeting and Mr. Armstrong, of the Western Fever Hospital, and Dr. Collie, of the Eastern Fever Hospital, for kindly providing me with scarlatinal tongues. Clinically I have to acknowledge the

services of a series of excellent clerks—Mr. Brushfield, Mr. Le Cronier, Mr. Sortain, Mr. Ogle, Mr. Drabble, and Mr. Barlow. And I have, finally, to thank the Fellows of the College and all who have formed my audience for the attention they have bestowed upon what I fear must too often have been tedious.

ON THE TREATMENT OF

CYSTIC GOÎTRE.

BY

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ON THE TREATMENT OF CYSTIC GOÎTRE.

My reason for bringing forward a method of treatment by no means new is, that, although it is mentioned in all the principal English and American works on Surgery, the prominence it deserves is not accorded to it, and, recently, cases have been published in which the treatment employed entailed serious risk to the patient, whose safety would not have been thus, unnecessarily, jeopardized had the measures I am about to describe been adopted.

The treatment which I advocate is that introduced by Sir Morell Mackenzie in a paper read before the Hunterian Society on January 22d, 1872, and published by him in the *Lancet* of May 11th, 1872 (vol. i., p 642).

His method, briefly, consists in tapping the cyst in its most dependent part, inserting a cannula, and when the fluid has been withdrawn, injecting into the cyst with a syringe a solution of perchloride of iron (two drachms of the salt to an ounce of water), then inserting a plug into the cannula, which has previously been secured in position, and allowing it to remain for seventy-two hours, after which time the plug is withdrawn, and not reintroduced if suppuration has taken place, poultices being applied, and the case treated as a chronic abscess.

But if, from the character of the discharge, it is seen that hæmorrhage is still taking place into the cavity of the sac, or that sufficient inflammation has not been established to cause a purulent flow, the cyst is to be again emptied, and the injection of perchloride of iron repeated at intervals of two or three days, until this latter condition has been produced.

The following conclusions, which Sir Morell Mackenzie published in 1875[1] as the result of his experience, are worthy of being remembered, and I will, therefore, mention them before describing his treatment in detail:—

"1. That any cystic goître which has attained the size of a hen's egg requires to be actively treated, even when the symptoms are not urgent.

"2. That smaller cysts which give rise to serious dyspnœa, or dysphagia, likewise require to be treated.

"3. That the conversion of the cyst into a chronic abscess, is the safest and most certain mode of treatment.

"4. That suppuration is best set up by injections of the perchloride of iron, as the disposition to hæmorrhage is thereby effectually controlled.

"5. That injections of iodine (in cystic goître) are dangerous, because often followed by sloughing.

"6. That there is a risk in the treatment by injections of iron, from the occurrence of too profuse suppuration, when the cyst has been allowed to attain too large size before treatment.

"7. That all operations on the neck are attended with the danger of air entering a vein and causing sudden death.

"8. That this risk is in proportion to the development of the veins and the propinquity of the tumor to the heart.

"9. That in pure cystic goître the chance of this occurrence is so slight, that it may be dismissed from consideration."

The duration of the treatment which I am about to describe is, generally, from three weeks to four months, according to the size of the cyst, the usual time being from six to eight weeks.

In dealing with a case of cystic goître, the place at which the trocar is to be introduced must first be decided upon. It is essential that the spot selected be at the most dependent part of the tumor to subsequently insure efficient drainage, upon which greatly depends the success of the operation, and it is an advantage to make the puncture where the cyst wall is thin, provided this condition is found in the position mentioned. Care must be taken to avoid a place traversed by a

[1] *Birmingham Medical Review*, vol. iv., p. 63.

large vein, and, when practicable, it is well to introduce the trocar as near the mesial line as possible. The spot having been selected, the skin should now be frozen by means of an ether spray, and when anæsthesia has thus been produced, a trocar with silver cannula attached should be passed through the wall of the cyst, the trocar being withdrawn as soon as the cavity is reached, and the cannula pushed further in by means of a blunt-pointed plug. For a large goître I select a cannula two inches in length, because I have found that if it is shorter it is apt to slip out of the cyst, in which case there is often difficulty in reintroducing it by means of the plug, in consequence either of the cyst wall having contracted, and, thereby, caused the hole in it to be no longer opposite the opening in the skin, or from the aperture in the cyst wall having closed. When this occurs it is necessary to again puncture with the trocar. For smaller goîtres a cannula of proportionate length should be employed. It is not necessary to use a very large cannula; the sizes I employ correspond to Nos. 7, 8, and 9, English catheter gauge. Those smaller than No. 7 are apt to become blocked with discharge, whereas, those larger than No. 9 are unnecessary in ordinary cases. The cannula having been inserted into the cyst, it should be secured in position by a tape passed round the neck. The plug should be then withdrawn, and the contents of the cyst allowed to escape, care being taken not to squeeze the cyst lest air should be sucked into its cavity. One or two drachms or more, according to the size of the cyst, of a solution of perchloride of iron (two drachms of the salt to an ounce of water) should be now injected into the cavity by means of the syringe, to be presently described, held vertically, and the plug introduced to prevent the solution from escaping. The reasons for using the aqueous solution of the salt, are, that it is more astringent than either the tincture or liquor ferri perchloridi (B.P.), and it is devoid of the free hydrochloric acid which they contain. The injection of perchloride of iron is made with a double object:—

First. To check the hemorrhage which takes place from the capillaries in the cyst wall on the fluid being withdrawn. This is a very important step, for, hitherto, the capillaries have been supported by the fluid contained in the cyst. That the hemorrhage results from the withdrawal of this support

is proved by the fact, that, although the fluid which first passes out of the cannula is of a pale albuminous character, yet, as soon as the cyst is emptied, pure blood is poured out.

Secondly. To produce sufficient inflammation in the cyst wall to destroy the secreting surface, and convert it into a granulating membrane.

The syringe used for this purpose has its nozzle fixed at an obtuse angle with the cylinder, the object of this arrangement being to allow any air which may be in the syringe, when held vertically, to rise to the top of the solution, and, therefore, render impossible the injection of air into a vein. This occurrence is further prevented by a button placed on the piston of the syringe above the cylinder, the button being screwed down for about an inch after the cylinder has been filled with the solution for injection, causing, by this means, the piston to be about an inch shorter than the cylinder, and, consequently, incapable of emptying it. The nozzle of the syringe is made to taper to the point, in order to allow it to accurately fit any cannula which may be used for this operation. The solution of iron having been injected into the cyst, it should be allowed to remain there for seventy-two hours. During this period the cyst will refill to a greater or less extent, and, if inflammation has been set up, it will be marked by pain and tenderness over the tumor, and there may, in addition, be signs of slight inflammation in its immediate neighborhood. Whilst the cyst is swelling, care must be taken to loosen the tape which holds the cannula, to a corresponding degree, otherwise the head of the cannula will be pressed against the skin and may cause it to slough; the tight tape will also greatly add to the patient's discomfort. During the after-treatment of the case, tightness of the tape may, generally, be considered to indicate distention of the cavity of the cyst, consequent upon the escape of pus being retarded by the cannula having become obstructed. If, on withdrawing the fluid at the end of seventy-two hours, it is found to contain blood, or to show no signs of suppuration, an injection of iron must again be made, and retained in the sac for seventy-two hours, as before, by means of the plug, this process being repeated until suppuration has been produced. Poultices should then be applied, and in some cases the plug may be permanently removed, but, usually, it is better to keep it in position for a few days, in

order to retain some pus in the cyst, and increase, thereby, the amount of inflammation. The quantity of pus, however, which is retained should not be large, and to insure this, the plug must be removed, several times during the day, to allow the discharge which has collected to escape. Whilst the plug is retained in the cannula the temperature generally ranges between 100° and 103° F., but quickly falls after the plug has been removed and the free exit of pus permitted; but, should it not do so (and from the character of the discharge it appears unnecessary to keep up the inflammation), the cyst should be washed out several times a day, by the injection through the cannula of tepid water containing some antiseptic.

Throughout the subsequent treatment of the case it is most important that thorough drainage of the cyst should be maintained; and it should be constantly borne in mind that pyrexia persisting after the plug has been removed from the cannula, or a sudden elevation of temperature during the later stages of the case, is almost invariably due to the cannula being blocked, and, consequently, causing retention of pus in the cavity of the cyst. With the view of preventing this occurrence I have, for some years past, used a piece of Ellis's drainage tube, which consists of silver or plated wire wound spirally to form a tube. I usually allow the turns of wire to remain touching one another, but, in some cases, it is well to slightly separate them, which is easily done by making gentle traction. To apply it, I select a piece of tubing which is a size smaller than the cannula, and consequently passes easily through it; and, having ascertained the precise length of the cannula by measuring the plug belonging to it, I introduce the tubing until it projects about half an inch beyond it into the cyst, and then secure it in that position by fastening its other end to the tape around the patient's neck. By this means, although the more solid portions of the discharge gather round the cannula and the piece of tubing which projects from it, and there remain, the fluid portion readily passes between the coils of wire into the lumen of the tube and thence away. To further insure efficient drainage it is necessary to wash out the cyst with tepid water, to which an antiseptic may be added, several times a day, and before doing so, to remove the Ellis's tubing. In some cases the mere injection of water into the cyst is not

sufficient to entirely remove all the thicker portion of the discharge, which, carried by the outward flow of the water, collects around the inner opening of the cannula, and blocks it. When this occurs, a fine probe, introduced through the cannula as soon as the syringe is withdrawn, and moved about so as to keep the end of the tube clear, will cause the previously obstructing pieces to come away with the water. It is useless to attempt to employ Ellis's tubing without a cannula, or to leave it long in position without being removed, because the granulations grow between the coils of wire and choke the lumen of its tube. Care must be taken to bend the inner cut extremity of the wire, so that it does not prick or scratch the wall of the cyst. Constant retention of a certain amount of pus may be caused, in this stage of the treatment, by the cannula projecting too far into the cyst, and, consequently, only allowing the escape of the overflow; the existence or not of this state of affairs can be easily determined by removing the cannula and noticing its color, the portion which projects into the cyst being always more darkly stained than that which is in contact with the tissues.

It sometimes happens that after a few weeks the discharge becomes thin, and no appreciable diminution in the size of the cyst takes place from week to week. This condition is usually produced by want of tone on the part of the patient, causing the granulations to become flabby, and the healing process, generally, less active. To remedy this, in addition to tonics and a more generous diet, it is well, after each time the cyst is washed out, to inject into it a solution of chloride of zinc (fifteen to thirty grains to an ounce of water), and allow the solution to escape through the cannula. This should be continued until, from the ready way in which the walls bleed when touched with a probe, and from other indications, it is seen that the granulations have reassumed a healthy aspect. A cannula should be retained in the cyst until its cavity is completely obliterated. Provided that efficient drainage is obtained through the cannula which was at first introduced, it is a mistake to remove it until the cyst has contracted around its inner extremity and the granulations have begun to block up the aperture. When this takes place the cannula should be replaced by one of the same gauge, but not more than a quarter of an inch shorter, which should likewise be retained

until the granulations begin to obstruct its inner orifice, when it should be replaced by a cannula again not more than a quarter of an inch shorter than the former one. A fresh cannula should be introduced on this system, from time to time, until the sinus made by the first cannula becomes too short to retain it, when the wound may be left to granulate.

It occasionally happens that during the after treatment the cannula is not well borne, in consequence either of its end impinging on the wall of the cyst and producing irritation, or causing discomfort by its pressure on adjoining parts. In these cases it is my custom to introduce an india-rubber cannula with the chance of its remedying this trouble. If the latter difficulty has to be met, I have, in several cases, found that relief was not obtained until the cyst had entirely closed, and a cannula had been dispensed with.

Sir Morell Mackenzie sometimes uses a silver cannula with a movable shield, which can be fixed at any part of it by means of a screw. In this way as the cyst contracts the tube can be shortened, and its length accurately adjusted to the requirements of the case. For patients who are not well off it is convenient, because it renders a number of tubes unnecessary; but it has the disadvantage of projecting further in front of the neck in proportion as it is withdrawn from the cyst, and this causes a little inconvenience in the arrangement of the dress, and the application of poultices. A multilocular cyst should be treated in the same way as a simple cyst; but if it is found that pus is retained in any one of the cavities, a cannula must be passed directly into that cavity at its most dependent part, and the same treatment adopted for it as is employed in the case of a simple cyst. A cyst containing a large amount of calcareous matter in its walls can only be effectually treated by excision; but this operation ought not to be performed unless the symptoms produced by the tumor demand surgical interference.

Before leaving this part of my subject, I may mention that Sir Morell Mackenzie, some years ago, employed a solution of tannic acid instead of iron, but the results were not so satisfactory as when the latter agent was used.

Not only does tannic acid fail to cause sufficient reaction—that is, to set up sufficient irritation—but it is apt to remain in the cyst cavity. In two of the cases in which I used it, the

powder formed a putty-like mass, which had to be dug out with a small spoon, introduced through the sinus made by the cannula, all other methods of removing it which were tried having failed to effect their purpose.

The patients whose history is now, briefly, given, and whose appearance, before and after treatment, is represented by a woodcut taken from a photograph, were shown at the Hunterian Society when this paper was read. Both cases were treated by the injection of the solution of perchloride of iron:

CASE 1.—Marie K——, aged thirty-four, single, first noticed a swelling in her neck twenty years ago. During the last eight years she has had a certain amount of dyspnœa whilst walking fast, or going upstairs. Her voice first became weak five or six years ago, and during the last four years it has remained much in its present condition. There is now paralysis of the left vocal cord, which is in the cadaveric position. The tumor has increased very much in size during the last three years, so much so, that for this period she has been unable to wear a collar. The goître had never been surgically treated before she came to the Throat Hospital. On admission, the circumference of her neck measured 19 inches. The cyst was tapped on December 16th, 1886, and twenty-one ounces of dark-brown fluid withdrawn.

CASE 2.—Mrs. C——, aged thirty-three, first noticed the enlargement in her neck nineteen years ago; the goître was then about the size of a hen's egg. For several years it did not increase much in size. About six years ago the swelling began to enlarge, and continued to increase slowly. She married three years ago, and was confined ten months afterwards, a marked increase in the size of the tumor being noticed after the birth of the child. Mrs. C—— states that during the last twelve months, when she has run downstairs, she has felt the fluid moving about "like water in a bottle." During the three months previous to her admission into the Throat Hospital, the increase in size was very great, so much so that she was obliged to enlarge the collar of her dress almost every Saturday, in order to enable her to wear the dress on the Sunday. The cyst was tapped on January 31st, 1887, and ten ounces and a half of bright-red fluid withdrawn.

CASE 1.

CASE 2.

Having detailed the treatment recommended by Sir Morell Mackenzie, I will now turn to some of the principal works on Surgery, with the view of ascertaining what assistance is to be derived from them by one having a case of cystic goître to deal with, and being uncertain as to the best course to pursue.

In the "System of Surgery," edited by Messrs. Holmes and Hulke, 1883, vol. iii., p. 594, is the following:

"The best treatment for cystic bronchocele is that introduced by Dr. Morell Mackenzie (*Lancet*, 1872), etc. etc. Simply tapping or incision has sometimes been successful, but there is always a danger of hemorrhage attending these measures, which is obviated by the use of the iron injection."

With these remarks I entirely agree.

Mr. Bryant, in his "Practice of Surgery," 4th edit. (1884), vol. i., p. 245, makes the following remarks:—

"Simply tapping a cerous cyst may cure it. When it fails, the cyst should be injected with half a drachm of a mixture of tincture of iodine and alcohol in equal parts; or, on this failing, of one of the same mixture and the liq. ferri perchloridi in equal parts. When these fail, a seton has been recommended, but the practice is dangerous, and should only be adopted when simple means are unavailing and further interference is requisite.

"In cysts of the isthmus, more particularly blood cysts, an incision into the cavity is a good and successful operation. Should a cyst after tapping suppurate, it must be dealt with as an abscess and freely opened as soon as the existence of pus can be made out, for the thyroid is a dangerous position for suppuration to occur. I have successfully treated one case of suppurating thyroid cyst after tapping by incision; but the cases in which this treatment is called for are rare. Dr. M. Mackenzie has (*Lancet*, May, 1872) advocated the practice of converting the cystic disease of the thyroid into a chronic abscess by the following means:—First empty the cyst," etc. etc.

Mr. Byrant's views, as expressed above, differ very materially from my own. He clearly recommends simply tapping the cyst as the first method to be employed, and only on its failure, the injection of iodine and alcohol to be adopted. Although it is perfectly true that "simply tapping a serous cyst may cure it," still, this result is the exception rather than

the rule, the usual consequence of such a proceeding being, that the cyst refills in the course of a few days, and not infrequently, before long, also increases in size. In addition to this, there is always the risk of hemorrhage from the capillaries rupturing on losing the support of the contents of the cyst; moreover, more than one death has been caused by simple tapping. For these reasons I do not recommend this treatment.

With the propriety of injecting tincture of iodine, I am also at issue with Mr. Bryant. Although cases have been cured by this method, still there have been so many in which severe inflammation of the cyst—extending to the cellular tissue of the neck, and causing extensive sloughing, and in some cases producing a fatal termination—has followed this method of treatment, that I consider it attended with considerable risk. The method advocated only aims at producing inflammation; it makes no provision for arresting hemorrhage, or providing an escape for pus, when suppuration has been established. To inject an irritant into a highly vascular cyst, placed in close proximity to the trachea and large vessels of the neck, with the view of causing inflammation—an operation which provides no exit for the escape of pus when it has formed—is, I consider, a highly dangerous proceeding.

With Mr. Bryant's remark that the treatment by seton is a dangerous practice, I entirely agree, but I am unable to accept his statement with regard to cysts of the isthmus.

This portion of the gland is a very frequent seat of cystic disease, some authors stating it to be the most frequent seat, and, as in many cases a cyst in this position attains a large size, I do not consider an incision into the cavity of a cyst so situated, in all cases likely to prove either a "good" or a "successful operation," whether its contents be serum or blood. Neither when a cyst suppurates after being tapped, do I advocate its being "freely opened as soon as the existence of pus can be made out," my opinion being that, if a cannula was not inserted in the first instance and left communicating with the sac, the sooner one is introduced the better. Also that the withdrawal of pus through this channel, and the subsequent washing out of the cyst with tepid water containing some antiseptic, through the same opening, is decidedly preferable to the treatment recommended, which is liable to be

followed by severe hemorrhage, and which, after all, only aims at accomplishing what the milder and safer method effects.

After the remarks of Mr. Bryant, which I have just discussed, he refers to Sir Morell Mackenzie's treatment; but from the order in which it is placed, and his preceding remarks, it is evident that he prefers the methods to which I have just given my grounds for objection.

Mr. Erichsen, in the "Science and Art of Surgery" (1884), vol. ii., p. 570, says:—"When the tumor is chiefly cystic, the fluid contents may be drawn off by tapping, and an endeavor may be made to cause the cysts to close by inducing inflammation in them by the injection of tincture of iodine. The fluid which flows from the cyst is often darkly stained with blood, and occasionally pure blood may escape in considerable quantities from the puncture.

"Morell Mackenzie advises the injection of the cyst with perchloride of iron to check the flow of blood that often takes place from the cyst wall. He recommends that the cyst be tapped with a trocar and cannula; after the fluid has escaped a solution of perchloride of iron (two drachms to an ounce of water) is injected, after which it may be withdrawn and a drainage tube substituted."

Mr. Erichsen, although alluding to Sir Morell Mackenzie's method, has sacrificed clearness for brevity, the result being that it is doubtful whether any one could carry out the treatment from the description which he has given of it. He places before it, also, the treatment by injection of tincture of iodine, to which I have already referred and given my grounds of objection.

Professor Gross, in his "System of Surgery" (1882), vol. ii., p. 356, says:—"For the cure of cystic goître, six methods of treatment are at the command of the surgeon: the seton, puncture, injections of iodine, incision, excision, and electrolysis; all more or less serviceable, but not one entirely free from danger." "Mere tapping of a cyst is not always free from danger," etc. "Injections of iodine occasionally succeed, but the operation is liable to be followed by severe inflammation, eventuating in rapid reaccumulation, and is in the main less certain than the use of a seton. When the cyst is very large previous tapping will be advantageous. From one to two drachms of equal parts of iodine and alcohol will be a suitable

quantity of fluid. If the water reaccumulates it must be let out by an early and free incision, and no effort must be spared to keep the inflammation within proper limits."

"Billroth, in 1877, reported thirty-five cases of cystic goître, in twenty-nine of which he effected a cure with the injection in each of four drachms of pure tincture of iodine. The operation was always followed by severe inflammation, and one case was fatal." "Incision of the morbid mass is occasionally practiced, and cases have been reported in which the operation was followed by marked diminution. The procedure is mainly adapted to tumors composed of large cysts, and the chances of success will be much increased if it be combined with the mopping of the affected cavities with iodine, and the use of a tent to provoke suppuration and granulation. Great care must be exercised, otherwise the free use of the knife will be attended with copious hemorrhage."

"Incision of the sac and uniting its walls with those of the skin, an operation which I believe originated with Von Bruns, has been performed eleven times by Billroth with three deaths."

My opinion with regard to the treatment by simply tapping, by injecting tincture of iodine, and, subsequently, making a free incision into the cyst, and by a seton, has been previously mentioned, and my views with reference to the advisability of attempting a cure by incision will be given presently, in connection with a case reported by Mr. Mayo Robson.

Professor Agnew, in his "Principles and Practice of Surgery" (1883), vol. iii., p. 497, enumerated in one list the treatment applicable to the various forms of goître, but did not specify for which variety of the disease the several methods mentioned were suitable, so that but little information as to his views on the subject can be obtained by any one previously unacquainted with the matter.

The above being the only information to be derived from the latest edition of the principal works on Surgery in this country and America, it is not surprising that considerable doubt as to which is the best method to adopt should arise in the mind of a surgeon called upon, for the first time, to treat a case of cystic goître, or that ordinary cases of this disease should be dealt with by exceptional measures, under the belief that all other methods are useless, or open to still more

serious objections than those known to apply to the operation selected.

In the *Lancet* of July 3d, 1886, is an account of a cystic goître treated by Dr. C. T. Vachell, of Cardiff, by "shelling out." The patient was a woman aged twenty-three, who had a tumor about the size of a cocoanut occupying the position of the right lobe of the thyroid gland and passing somewhat over the middle line of the neck. Fluctuation could be felt all over the tumor. Neither breathing nor swallowing was seriously interfered with. About three weeks before admission a teacupful of bloody serous fluid had been withdrawn by a trocar and cannula, and the tumor, in consequence, considerably reduced in size, but reaccumulation took place in a day or two, and in less than six weeks the tumor had attained a larger size than before, the circumference of the neck being 17¾ in. The patient was informed that if the tumor remained serious results might ensue, and she consented to an operation. Ether having been administered, a vertical incision 2½ in. in length was made through the skin over the most prominent part of the tumor, and at once the surface of it came into view. "Up to this point very little hemorrhage had occurred. Without much difficulty the tumor was now shelled out, but the hemorrhage which commenced with this process and continued throughout was very profuse. Firm pressure was maintained by an assistant, the surface was mopped with very hot water mixed with tincture of iodine (1 in 20), and as bit by bit the surface was exposed all the bleeding points were ligatured. The edges were then brought together, and a drainage tube inserted." "The tumor, which was irregularly ovoid in shape, measured 13 in. by 9 in. and weighed twenty ounces. On being cut into, nine ounces of dark sanguineous fluid escaped, similar to what had been removed by tapping, but darker."

The patient left the infirmary twelve days after the operation.

Although the operation was most skillfully performed, and the result of the treatment as far as reported quite successful, still, I consider, from the account given of the case, that there was nothing to justify the measures which were adopted. The case was apparently an ordinary one of cystic goître, and it is distinctly stated that neither breathing nor swallowing was

seriously interfered with. That the cyst would refill after being tapped, might have been foretold, it being the exception when this does not occur; also that it would be liable to attain a greater size, might have been stated with equal confidence. These usual results, however, having followed the tapping, Dr. Vachell appears to have thought that no course was open to him but to excise the portion of the gland containing the cyst. Although in the case just recorded the operation was successful, its performance is always attended with a considerable amount of risk to the patient. More than one death has resulted from hemorrhage, which, as it was in this case, is frequently very profuse, and the evil effects which may arise from the excision of large portions of the thyroid gland are too well known to need comment. After the grave consequences which are liable to attend the operation, the mere question of the size of the scar which results sinks into insignificance, although, I hold, that in the surgical treatment of exposed portions of the body this point ought to be considered.

In the *Lancet* of January 22d, 1887, p. 174, is the report of a paper which Mr. Mayo Robson, of Leeds, read before the Clinical Society, in which he advocated the treatment of thyroid cysts by antiseptic incision, stitching the edge of the cyst to the skin, and scraping the interior of the cyst sufficiently to separate all colloid material, then draining for a short time under an antiseptic dressing, and afterwards packing with zinc lotion and lint. Mr. Mayo Robson thought that the advantages of this method were its simplicity, safety, and certainty. He stated that he would not again practice complete excision, having had one case in which asphyxia and another in which hemorrhage caused great anxiety. The advantage claimed by Mr. Mayo Robson of stitching the cyst wall to the skin, was that by this means the cyst was shut off from the surrounding cellular tissue, and, consequently, the extension of inflammatory processes was prevented.

The elaboration of the treatment by incision, just referred to, presents no novel feature, as the extract which I have quoted from Gross's "System of Surgery" clearly shows. The fact that three deaths occurred in the eleven cases operated upon by Billroth does not offer much encouragement for the adoption of this method of treatment, especially if he did

not scrape the interior of the sac, which proceeding must add considerably to the risks of it. The object of the treatment advised by Mr. Mayo Robson is the same as that of Sir Morell Mackenzie—viz., the destruction of the secreting surface of the cyst and subsequent union of its walls; but the means which he takes to produce this result compare most unfavorably with those which I advocate.

Before the secreting surface can be destroyed it must be reached. Mr. Mayo Robson effects this by an incision, Sir Morell Mackenzie by a puncture. The former method is always attended with hemorrhage, which in many cases is very profuse; the latter is bloodless, because the pressure produced by the cannula plugs any vessel that may have been wounded, except in those rare cases in which a large vessel in the interior of the cyst becomes injured, and in these, no serious result need be feared, because but little, if any escape of blood can take place externally, by the side of the cannula, and the amount of flow internally is limited by the size of the cyst, even if pressure and ice-bags have failed to check it previously. In order to destroy the secreting surface, Mr. Mayo Robson advocates scraping the wall of the cyst sufficiently to separate all colloid material, and subsequently packing the cyst with zinc lotion and lint. Sir M. Mackenzie recommends a solution of perchloride of iron to be injected into the cavity, with the twofold object of checking the hemorrhage from the cyst walls which follows the withdrawal of the fluid contents, and, subsequently, of producing sufficient inflammation to destroy the secreting surface. Bearing in mind the extreme vascularity of the cyst wall, and the delicate structure of the vessels, I consider Mr. Robson's method of destroying the secreting surface to be highly objectionable, even if not positively dangerous. The steps of the operation recommended by Mr. Robson, cannot be performed without producing hemorrhage, which is always liable to be profuse; while, on the contrary, the object of the treatment which I advise, throughout, is to prevent hemorrhage. Holding the views, above stated, with regard to the method of treatment advocated by Mr. Mayo Robson, I am unable to consider it either simple, safe, or certain.

Having been accustomed, myself, to treat cases of cystic goître by Sir M. Mackenzie's method, and to see the same

course adopted by others, I was unaware, till recently, of the methods of dealing with this disease advocated in the last editions of the leading works on Surgery, having had no occasion to refer to them on this subject, being well satisfied with the plan I employed, and knowing how disastrous many of the older modes of treatment had proved. It was not, indeed, until my attention was called to the matter by the case referred to, published by Dr. Vachell last year, that I looked at the most recent English and American works on Surgery, to see what treatment their authors recommended. Had I done so, being ignorant of the best course to pursue, I confess that when I had finished such study I should have been a sadder but not a wiser man, because no assistance would have been obtained, notwithstanding that the five leading works had been consulted. I should have found that no two authors agreed upon what is the best method to adopt, and that, in fact, all recommend a different proceeding My efforts to obtain advice would have shown me that Messrs. Holmes and Hulke advocated Sir Morell Mackenzie's method; that Mr. Bryant recommended puncture as the first means to be adopted; that Mr. Erichsen advised the fluid contents to be drawn off by tapping, and an endeavor made to cause the cyst to close by inducing inflammation by means of an injection of iodine; that Professor Gross stated that six methods of treatment were at the command of the surgeon, all more or less serviceable, but not one entirely free from danger; and that Professor Agnew, apparently, did not consider the subject of sufficient importance to necessitate a separate paragraph being given to it, he having enumerated the various methods of treatment applicable to the several forms of goître in one list, and in such a manner that only a person possessing a full and accurate knowledge of the subject could distinguish which method was suitable to a particular form of the disease. A closer examination of the treatment recommended would have shown me that in two of the three English works injection of tincture of iodine was favorably spoken of, but under different conditions; Mr. Erichsen having advised the cyst to be emptied before the iodine was injected, whereas Mr. Bryant advocated the injection of tincture of iodine and alcohol in equal parts, without any allusion to the preliminary measures recommended by Mr. Erichsen. Professor Gross, on the other hand, whilst

stating that injections of iodine occasionally succeed, went on to say that the operation was liable to be followed by severe inflammation,—eventuating in rapid reaccumulation,—and was in the main less certain than the use of a seton; whereas Mr. Bryant stated that this latter method was dangerous, and should only be adopted when simple means were unavailing and further interference necessary. With such diversity of opinion it would have been extremely difficult for me to come to a conclusion as to the best course to adopt, especially as the weight of opinion was against the treatment which seemed most rational; and I consider that had I selected an operation which exposed my patient to unnecessary risk, the responsibility for the course adopted would have rested on the shoulders of most of the authors of the surgical works which deal with this disease rather than on my own.

Bearing in mind the frequency with which cases of cystic goître are met with, and the large number of deaths which have followed the attempts to effect a cure, I maintain that the treatment of this disease demands more serious attention than surgeons have hitherto accorded it.

In conclusion, I will say that if I have clearly demonstrated the decided advantages possessed by Mackenzie's method, as compared with the other modes of treatment which have been employed, and this should lead surgeons to adopt it, and abandon, as more dangerous, the other operations which have been performed, I shall consider that the time which I have devoted to ascertaining the views of other men on this subject, submitting them to critical examination, and testing them by the light of experience, has been well spent, and the object aimed at in this paper fully attained.

NEW REMEDIES:

The Principal Medicinal Agents Introduced into the Materia Medica

FROM 1878 TO 1888.

BY

DR. C. CAUQUIL,

Late Interne of the Hospitals of Paris, France.

INTRODUCTION.

THE aim of this work is to fulfil a want which becomes more evident every day. Of late years therapeutic researches have multiplied in unheard-of proportions, and the number of new medicines proposed, experimented with or introduced into practice, comprises already a considerable list.

There is not in France, if we may so speak, a medical practitioner who does not seek to have his own remedy or to discover new properties in medicines proposed by others.

From this collection of therapeutic novelties, which the medical press discloses day by day, there results confusion, a veritable chaos. Indeed, often violent disputes arise on the subject of the effects or the rational use of one or another agent, and in the short space of a few months we witness the triumphal rise of a sovereign remedy and its fall into absolute neglect.

Amid these numberless discussions, the practitioner remains sceptical, and he refrains from the use of a really valuable agent. Or, if he would test recent therapeutics, he is necessarily condemned to search many medical publications for the scattered data which could guide him on his way. Hence great weariness and loss of time.

We have therefore thought it useful to condense into one volume all that has been published for ten years in the line of therapeutic discoveries; to analyze succinctly the works of our celebrated physicians, especially those of the society of therapeutics; finally, to reject the medicines known to be

useless or less efficacious than their already existing successors, and then to state the most common formulæ or the most convenient methods of administering the new medicines; this has been our design.

In conclusion, we would say that since we note the very different properties of each agent (for example: antipyrin, both antipyretic and a depressor of nervous activity), we have thought it better to lay aside classification of the different agents and to follow in this work only alphabetical order.

DR. CAUQUIL.

NEW REMEDIES.

ACETANILID.

ALSO called phenylacetamid; formula, C^8H^9N. A white substance, crystalline, inodorous, of generally tart taste; soluble in alcohol, slightly soluble in cold water. Its discovery is due to Gerhardt.

Acetanilid was first used at the clinic of Kussmaul in Strasburg. Soon afterwards, Lépine of Lyons and his pupils made interesting researches into the physiological and therapeutical effects of this substance; and, finally, Dujardin-Beaumetz and his pupil Weill, by recent experiments, rendered the more valuable by the extreme precision with which they were conducted, definitely introduced acetanilid into our materia medica. The publications of these two experimenters are above all what we propose to summarize in this section.

Physiological Effects.—In poisonous doses acetanilid produces in animals the following successive effects: stupor, prostration and hesitation in movements, accompanied by great and rapid decrease in temperature.

Respiration decreases progressively, and, strangely enough, sensibility is rather quickly abolished in the posterior portions of the body, while it continues more acute in the anterior parts; analgesia and collapse become more and more marked, and death ensues after a series of convulsive shocks succeeding each other at very brief intervals. In short, acetanilid acts principally upon the circulation and the nervous system.

Action on the Circulation.—Shortly after the administration of acetanilid, there is found manifest acceleration of the cardiac pulsations, which at the same time become more energetic; later they slacken and grow irregular. Similar modifi-

cations are observed in the intravascular tension, increas followed by diminutio

The state of the blood is considerably modified; the propor tion of oxyhemoglobin diminishes and methemoglobin is forme in notable quantity, whence the lowering of temperature pro duced by large doses; it is thus that in the rabbit Weill sav the thermometer fall eight and even ten degrees below th normal.

Action on the Nervous System.—Unless administered i poisonous doses, acetanilid does not act upon the brain; thi is not the case, however, in regard to the spinal marrow an the bulb; in fact the reflexes are notably weakened, and pneu mogastric excitability is diminished.

In reality, then, the action of acetanilid upon the cord show itself in phenomena of collapse, which predominate in the ani mal in cases where physiological effects of some intensity ar reached, and by the depression of cardiac and respiratory functions well displayed by graphic tracings.

Acetanilid acts upon the peripheral nervous system by di minishing sensibility and producing vaso-motor disturbances which may perhaps be connected with the action produced o the bulb where the vaso-motor centre and the thermic centr are localized.

Therapeutics.—Of acetanilid as an antipyretic Dujardin-Beaumetz says:

"As an antipyretic, acetanilid is a very mediocre medicine, which lowers the temperature by acting both upon the nervous system and the circulatory force of the blood. Moreover, its antipyretic action is unequal; in small doses it has been known to produce considerable thermic depression; it finally leads to cyanosis. Acetanilid, then, has been shown to be an antipyretic greatly inferior to antipyrin; it rather resembles phenic acid in its physiological action. So, in my opinion, acetanilid as an antipyretic ought to be rejected from therapeutics for the same reason that phenic acid has been rejected."

But as a nervine acetanilid is, on the other hand, an acquisition of great value to therapeutics. The first thing to be noted is the harmlessness of small doses upon the healthy man, while upon the febrile patient the small quantity of 7½ grains may cause, through great lowering of the temperature, cyanosis and dangerous collapse.

The researches of D. Beaumetz have borne upon the action of acetanilid against (1) pains in general, (2) special pains in tabetic patients, (3) epilepsy.

In the first category acetanilid has been found inferior to aconite in the treatment of essential facial neuralgia, but when this neuralgia is due to change in nerve fibre, as in optic neurosis, acetanilid is superior to every other medicine; thus a man suffering from great peri-orbital pains was cured after the administration of gr. xxijss of the substance, although for months aconite, bromine and iodine had been ineffectual.

In rheumatic pains, acetanilid has proved as efficacious as salicylic acid, over which it has at least the advantage of not provoking cerebral troubles.

In tabetic patients sharp pains are happily modified by acetanilid. The same effects have been observed in the clinic of Grasset of Montpelier; as long as the medicine was continued the pains did not appear; they returned as soon as the use of acetanilid was abandoned.

Some ataxic patients have resisted the action of the medicine, and others, after temporary relief, have had a return of their pains; there can be no doubt that this is due to the diversity in the origin of the sharp pains of the tabetic patient.

Concerning epilepsy, amid a number of cases of amelioration, D. Beaumetz cites that of a child twelve years old, which sufficiently demonstrates the effects:

Epilepsy had lasted for several years; a medication of bromine was first instituted and led to a slight alleviation without causing the disappearance of the attacks, which recurred every eight or fifteen days; the doses of bromide were increased, but intense phenomena of bromism made it necessary to abandon this medicine. Dujardin-Beaumetz then bethought himself of acetanilid, which he ordered in doses of gr. vij three times a day, and three months afterwards the attacks had not reappeared.

Encouraged by this report, published in March, 1887, I prescribed acetanilid for an old lady who had epilepsy for several years; paraldehyde, after having given her a respite for about two months, proved powerless to prevent the attacks, which recurred at least once a month. I prescribed acetanilid in solution in elixir of Garus, the dose being that established by Dujardin-Beaumetz; to avoid habit, I continued the medi-

cation for fifteen successive days, and I ceased all treatment the fifteen days following; under the influence of this medication the patient remained ninety-seven days without a repetition of the attacks; but they returned anew although at somewhat longer intervals.

In connection with these facts it is proper to note two cases of Grasset of Montpellier in which acetanilid failed utterly.

To sum up: acetanilid, given in doses of from 15 to 30 grains to persons who are not feverish, is attended with no inconvenience and may be continued for months.

In feverish patients it rapidly lowers the temperature, and unless prudently employed it is attended with danger of unexpected cyanosis and collapse; consequently as an antipyretic it ought to yield to kairin, to thallin and to antipyrin.

As a nervine acetanilid seems to apply especially:

To rheumatic pains, muscular and articular.

To neuralgic pains due to alteration or compression of the nerves.

To the sharp pains of tabetic patients.

Finally, to epileptic attacks.

Method of Administration.—We have already said that to be active a dose of acetanilid should reach gr. vijss without exceeding from 30 to 45 grains.

This substance being almost insoluble in water, we may choose between two methods of administering it·

(1.) In wafers.

(2.) In solution in an alcoholic liquid.

Let us add that it is always beneficial to divide the doses, and to give the total quantity in three portions during the day.

ANTIPYRIN.

Antipyrin belongs to the class of phenols; it is dimethyloxyquinizine, and its formula seems to be $C^{22}H^{12}N^{2}O^{2}$.

First experimented with by Filehne (of Erlangen), who made known its antipyretic properties, and for this reason introduced it under the name of antipyrin, it quickly spread in the clinics of Germany, Belgium and Italy, before being admitted into the French hospitals, where its general use was

established by the experiments of M. M. Huchard, Jaccoud, Dujardin-Beaumetz, G. Sée, etc.

Antipyrin appears in the form of a brilliant, yellowish-white, crystalline powder. It is fusible at 113 degrees, almost inodorous, having a bitter taste. It is hardly soluble in ether; alcohol, chloroform and water dissolve it well.

Its most palpable reaction is the reddish-brown precipitate which it gives in the presence of iodide of potassium.

Pharmacodynamics.—The physiological and therapeutical effects of antipyrin are absolutely united; from a practical point of view it is above all necessary to consider them from two stand-points: antipyretic action and analgesic action.

Calorification.—In the healthy man the thermic modifications produced by antipyrin are in every case inconsiderable; it is not so, however, with the feverish person, and here the lowering of the temperature is a phenomenon which has always been observed, although with variations in the rapidity of the thermometrical fall and in the duration of the antipyretic action.

German doctors, experimenting with large doses (75 to 90 grains in three portions at intervals of an hour), ascertained a thermometrical fall at the end of a couple of hours, a fall which reached its maximum at the end of from four to five hours and lasted from six to twenty hours, after which a gradual rise took place.

In France experiments have been made with very small doses, from 7½ to 11 grains, which produced a lowering of temperature at the end of two hours; this state may be maintained by repeating the same dose every two hours up to forty-eight hours. The lowering of temperature varies from one to three degrees.

Professor Jaccoud has above all insisted upon the transient action of antipyrin, and the necessity of administering it in frequently repeated doses without having recourse to large doses.

The child bears antipyrin well, the dose varying with the age.

Indications.—By reason of its antipyretic action, antipyrin is indicated in all maladies where there is continuous fever; but it must not be forgotten that its action is transient, and that if it diminishes fever, with the greater part of the incon-

veniences which the latter brings, it remains absolutely ineffectual against the disease itself; and that it is necessary to protect the system against a sub-normal fall of temperature which might become dangerous.

The hectic fever of consumptives is of all indications the most explicit; therein lies the real triumph of antipyrin. By its use sweats are avoided, weariness, insomnia, acceleration of the respiratory rhythm are diminished, and the patient feels a sense of life which is often prolonged several days after the administration of the drug.

In typhoid fever and erysipelas rather large and often repeated doses are necessary, while less quantities suffice in rheumatic fever, the beginning of eruptive fevers or ephemeral fevers.

Method of Administration.—Filehne, and with him the German doctors, gives gr. xxx of antipyrin shortly before the access of fever and repeats this dose twice at intervals of four hours. In consumptives it is always well to reduce this dose to gr. xv. In France, Dujardin-Beaumetz and Huchard do not go beyond gr. viiss and Daremberg, after having given gr. xv of the substance, repeats this dose whenever the thermometrical rise attains 0.3° C.

Bouveret gives a first dose of gr. xv, then successively doses of gr. viiss, according to the intensity of the fever, but without exceeding gr. xlv during the day. As is easily seen, the difference between these two modes of administration is slight.

The stomach is generally preferred for the absorption of antipyrin; the latter may be taken in solution or in wafers; in the last case it is well for the patient to swallow a certain quantity of liquid immediately after the ingestion of the remedy.

Hypodermic injections are not without inconvenience (pain, abscess), and administered by the rectum the medicine is deprived of part of its efficacy.

In children, Demme prudently advises that the dose of gr. iij be not exceeded during early infancy, and that of gr. viiss up to five years of age.

Phenomena of Pain.—The discovery of the analgesic action of antipyrin is almost exclusively due to Professor G. Sée, who clearly established the fact in three successive communications to the Academy of Medicine during the year 1887.

In a first series of subjects affected by apyretic rheumatism, with or without hydrarthrosis, who had been treated without success by means of salicylate of soda and the actual cautery, the pain disappeared within a few days and did not return so long as care was taken to continue the drug in small doses for about a week. The same effects were observed in attacks of acute gout with uretic deposits; antipyrin, in doses of from ʒ j to ʒ jss, controlled the pain and the articular swelling in from two to four days.

Antipyrin has been found a sovereign remedy for affections of the sensory nerves; it has rarely failed in headache, whether cephalalgia of long standing, sick headache or facial neuralgia.

It has also relieved peripheral neuralgia and the serious neuroses due to diabetes or zona, and even in tabetic patients it has not failed to calm the violent pains attending the beginning of ataxy.

Antipyrin has soothed the severe pains of cardiac and aneurismal subjects.

Doses.—The requisite dose of antipyrin has been in every case not less than gr. xlv and not more than ʒ jss; gr. xv in half a tumbler of cold water being administered every two or three hours. The most serious inconvenience resulting from the use of this drug is a stinging eruption, which ceases, however, as soon as the doses are reduced.

In view of the successes he has obtained, Professor G. Sée does not hesitate to pronounce that antipyrin ought to be substituted for morphine, even in hypodermic injections; the dose of gr. viiss he deems sufficient to replace injections of morphine in all their indications. "To be sure," says he, "those who have acquired the morphine habit will not be content with antipyrin, which does not produce the sensations and the intoxication so much desired by invalids; but it quiets pain suddenly, it diminishes at once the reflex excitability of the spinal marrow—that is to say, the vague, general, nervo-muscular pains so often productive of hysteria or neurosis. Henceforth antipyrin will take the place of morphine, and will save from the opium habit those who at any price seek this remedy for their pain.

Secondary Effects.—Before leaving the subject of antipyrin, let us note the several altogether secondary effects produced by this drug upon the organism.

It only slightly affects the pulse and the arterial tension; indeed this is a point so disputed by different experimenters that new studies are needed to elucidate it.

The hemostatic action of antipyrin by means of local applications has been proved by Hénocque, but has not been generally utilized.

Antipyrin does not act upon the respiratory rhythm except where the latter has been pathologically exaggerated by the fact of thermic rise, and then it diminishes its frequency by combating excessive rise of temperature.

On the ingestion of the drug, phenomena of gastric intolerance have been noted, but they have occurred chiefly in consumptive subjects.

Antipyrin acts upon the urinary apparatus by diminishing the quantity of urine and the proportion of urea, facts which should be considered in cases of renal affections.

To the appearance of the exanthema already noted, it is proper to add the increase of perspiration first remarked by Filehne.

Dujardin-Beaumetz and Debove, and then Bouveret, Lépine, etc., have observed a weariness, a vague uneasiness which, in certain subjects, can extend to collapse; Jaccoud has cited examples of this nature, and these cases are valuable in that they are the result of a particular idiosyncrasy and not of the doses administered.

To sum up: antipyrin is a good transient antipyretic, and above all a powerful analgesic, and the slight inconveniences it involves should not prevent its ranking among our best therapeutic agents.

M. Caravias has still further extended the domain of antipyrin by utilizing it in blenorrhagia in the form of urethral injections in solution of one-tenth part, or by incorporation in medicated gelatine bougies, the surface of which has been steeped in a concentrated solution of antipyrin, and which should melt in the urethra. The results seem to have been satisfactory.

In conclusion, let us note some more applications which, in various diseases, may be made of antipyrin combined with other medicines:

With Bromide of Potassium in Epilepsy as also in Nervous Subjects (Prof. Sée).

Antipyrin, ...	gr. xxx to xlv.
Bromide of potassium, . .	gr. xlv to ʒ j.

With the Iodide in Cardiac Subjects.

Antipyrin,	
Iodide of potassium, . . .	āā gr. xv to xxx.

With Tincture of Iodine and Iodide of Potassium as a Gargle.

Water, ...	℥ j.
Iodide of potassium, ...	gr. xv.
Tincture of iodine, ...	gtt. xxx.
Antipyrin, ...	gr. xxx.

With Naphthalin, Iodoform and Tannin, for Colic.

Naphthalin, ...	gr. ¾.
Iodoform, ...	gr. ⅓.
Tannin, ...	gr. iss.
Antipyrin, ...	gr. iss.

In a Pill.

3 or 4 of these pills in succession.

Finally, there is undoubtedly a series of other medicinal combinations, many of which remain to be formulated and called upon for service.

Balm of Gurjun.

The balm of Gurjun, or oil of wood, known in France since 1838, was formerly used by the physicians of India as a cure for blenorrhagia, but it was introduced into our pharmacopeia only in 1876, under the auspices of Vidal, who employed it in blenorrhagia; Mauriac, C. Paul, and Malez used it successively in the same cases.

The balm of gurjun is a resinous oil furnished by many species of the genus Dipterocarpus of the family of Dipterocarpæ. It is a thick, viscous, cloudy liquid, in odor resembling copaiba; bitter and aromatic in taste.

Physiological Effects.—In does of from ʒ j to ʒ j gr. xv the gurjun is perfectly tolerated; its taste is less disagreeable than that of copaiba; in the stomach it provokes a sensation of heat in the epigastric region, some eructations, but rarely nausea and vomiting. Its action upon the intestines is manifested by one or two sudden stools two hours after ingestion. If the dose is increased, say to gr. Cv or ʒ ij, signs of gastro-intestinal intolerance appear: vomiting, colic, diarrhea. Elimination takes place in different ways: the essence is eliminated by perspiration and pulmonary exhalation, while the resin passes off by the urine, to which it communicates a slight balsamic odor analogous to that of turpentine.

Therapeutics.—Among the cutaneous affections which have been relieved by the balm of gurjun, the first in importance is leprosy; Dougall, who was the pioneer in this respect, caused the bodies of the patients to be rubbed two hours twice a day with a mixture of

Balm of gurjun, ...	1 part.
Aq. calc., ...	3 parts.

The improvement in most cases was very marked. In 1876 Vidal, at the Hospital St. Louis, obtained almost complete success in a case of tuberculous leprosy by means of friction, repeated morning and evening, with a mixture of balm of gurjun and lime water in equal parts, accompanied by the daily swallowing of from ʒ j to gr. Cv of balm. This case, added to the observations of the East Indian and English doctors, tends to establish the balm of gurjun as a good auxiliary if not an heroic remedy for leprosy.

Other skin diseases, chronic eczema, lupus, tubercles of the skin, if we may credit certain writers, have been happily modified by the balm of gurjun, but confirmation of these results is yet wanting.

As an anti-blenorrhagic, the balm of gurjun is better known; a number of physicians praise it, and Vidal, the chief experimenter with the medicine, tells us that mild cases are cured by it in from six to eight days, and that discharges treated in the beginning are cured in from fifteen to twenty days.

In prostatitis and vaginitis the balm of gurjun has given quick results.

Method of Administration.—The preferable form of administering the medicine is in a potion according to one of the following formulæ:

Formula of Vidal.

Gurjun,	
Acaceæ, ...	āā ʒ j.
Syr. simpl., ...	ʒ iij.
Infusio badian., ...	℥ j ʒ ij.

To be taken twice a day before meals.

Formula of Mauriac.

Gurjun, ...	℥ ss.
Acaceæ, ...	ʒ iiss.
Syr. simpl., ...	ʒ viiss.
Aq. menth. pip., ...	℥ j ʒ ivss.

Three times a day.

The second of these prescriptions tends to produce gastro-intestinal troubles, and the first seems to us preferable. A glass of Malaga wine may be given after each dose, according to the advice of Vidal.

For external use, we may have a liniment composed of the balm and lime-water in equal parts; Vidal has used this preparation as a remedy for vaginitis; he has employed it locally with the aid of a tampon of cotton which he kept in position by means of a second dry tampon. A speedy cure has ensued in every case.

Boldo.

Boldo or boldu is the name commonly given to the young shoots of the pneumus-boldus of the family of Monimiaceæ. Native of Chili. The analysis of the plant has discovered, as its particular characteristics, an abundant essence, chiefly in the leaves, and a bitter principle regarded as an alkaloid and designated by the name of boldin.

Physiological Effects.—Studied at first by Dujardin-Beaumetz, these effects have been successively sought for by Verne (1882) and Laborde (1885).

As to the digestive apparatus, the tincture of boldo, in doses of gr. xv in a tumbler of water, produces an aromatic sensation to the taste, then heat in the epigastric region, and an excitation of the digestive functions. If the dose exceeds gr. xxx the heat becomes very painful, and it is followed by sudden colic, diarrhea and vomiting. The essence causes the same effects if it is given in doses exceeding gr. vj.

The influence of boldo upon the circulation is insignificant, and if it exists at all, new experiments are required to prove it.

The action of boldo upon the nervous system is manifested in drowsiness, more or less marked according to the dose. Subcutaneous injections of boldin produce the same effect, and moreover insensibility at the point of injection; and the infusion into the eye of a few drops of a solution of boldin causes insensibility of that organ, just as cocaine does.

The elimination of boldo and boldin takes place through the urinary channels, but without increase in the quantity of urine; only the proportion of urea is augmented.

Therapeutics.—Of the several properties of boldo, use has been made only of its power to excite the digestive and to stimulate the urinary functions; as to the hypnosis produced by the drug, it has not yet been tested.

Dujardin-Beaumetz has used boldo in cases of atony of the stomach, with or without general debility, and it is useful in simple anemia, in dyspepsia and convalescence from long and depressing illnesses.

Delioux of Savignac assigns to boldo an important part in the treatment of affections of the urinary passages. We have said that boldo possesses no diuretic power, but its essence creates in the mucous membranes by which it is eliminated an action quite comparable to that exercised by copaiba and turpentine; catarrh of the bladder, either acute or chronic, has also been successfully treated by boldo.

Mode of Administration.—The pharmaceutical preparations used are the tincture, the essence, the alcoholic or aqueous extract, the wine, the syrup and the elixir.

The essence of boldo is given in doses of from gr. iij to vj, and, because of its burning taste and sharp odor, in the form of pills.

The tincture may be taken in a potion, in doses of from gr. viij to xv. As to the wine and the elixir, it is well to prescribe

them by the wineglassful after each meal; the last preparations are the most acceptable to the sick.

Bromide of Ethyl.

Bromide of ethyl, or bromohydric ether, discovered in 1829 by Serullas. It is a colorless, slightly odorous liquid, at first sharp then warm in taste; its formula is, C^4H^5Br.

First utilized abroad, particularly in the United States, it has been studied latterly in France by Rabuteau, Terrillon, Bourneville, Roux, Ducasse, etc., and they consider it an anesthetic worthy of comparison with chloroform and ether.

Physiological Effects.—The spray of bromide of ethyl upon the skin produces local cooling; the tegument grows rapidly pale and the lowering of temperature can be pushed far enough to lead to the formation of an erosion.

Provided it is perfectly pure, the bromide of ethyl produces no ill effects upon the respiratory passages.

Neither does the digestive tube appear to be incommoded by the ingestion of the drug; Rabuteau has succeeded in administering as much as 18¾ grains without other effect than a slight warmth in the epigastric region.

Without speaking of the anesthetic action wrought upon animals by the bromide of ethyl, we state the opinion of Terillon, who, in accord with the American surgeons, inclines to the belief that in man the bromide of ethyl might be a more rapid and less dangerous anesthetic than chloroform.

According to this surgeon, the period of excitement should be less and be succeeded by little muscular rigidity, rarely tonic convulsions, and, in the majority of cases, a congestion of the face, very pronounced in alcoholic subjects. Anesthesia should be produced at the end of from two to three minutes of inhalation.

Bromide of ethyl has been charged with producing exaggerated cyanosis, pupillary dilatation, smallness of pulse, threatenings of asphyxia and too often incomplete anesthesia.

The vomiting, easy to arrest in the beginning by augmenting the dose of the inhalations, is in fact as frequent as with chloroform, if we take into account cases where it occurs on awakening and even a certain time after the anesthesia.

The elimination of bromide of ethyl takes place wholly by way of the respiratory organs.

Therapeutics.—As a local anesthetic the bromide of ethyl is incontestibly superior to similar agents, owing to the following properties:

Not being an irritant it can be applied to a bleeding wound without producing smarting. Almost inodorous, it does not inconvenience either the operator or his patient. Not being inflammable, it allows the employment of the cautery without fear of producing a burn by inflammation of the pulverized vapors.

These qualities have caused it to be used by the principal surgeons in most of the cases where local anesthesia was indicated.

The criticisms noted above have prevented the preference of bromide of ethyl to chloroform, which gives complete muscular resolution and an anesthesia as perfect as could be desired.

In medicine, Bourneville and d'Ollier have used bromide of ethyl for epilepsy, in the form of inhalations; the result of their experience leads them to the conclusion that it constantly diminishes the attacks without radically curing the disease.

The same physicians have obtained the cessation of convulsive-hysterical phenomena by the aid of the same means.

Rabuteau advises a trial of the bromide of ethyl in gastric pains and whooping-cough.

In obstetrics, the bromide of ethyl, extolled by the Americans, has not in the hands of the French doctors produced encouraging results. According to Ducasse, the inhalations of this gas retard delivery by diminishing the energy of the uterus without completely doing away with the pains.

Mode of Administration.—For local anesthesia, Terillon advises the use of an atomizer with large tube held at 3¾ inches from the field of operation; anesthesia takes place in two or three minutes.

Inhalations are made as with chloroform, but to produce the maximum effect, the agent should be abundant from the outset.

Taken internally in the form of pills or of syrup, the bromide of ethyl may be given in doses of from 15 to 30 grains.

Incorporated in a liniment it may be used externally in the same manner as chloroform or ether.

Cascara Sagrada.

This is the name given to the bark of the *Rhamnus purshiana,* plant of the family Rhamnaceæ.

First experimented with in the United States by Bundy, Pearse, etc., the cascara sagrada made its appearance in France under the auspices of Landowski, and has been particularly studied in the Hospital Cochin under the direction of Dujardin-Beaumetz.

The analysis of this bark has given a fixed oil, a volatile oil, and a crystalline compound yet ill defined.

It is employed only in the shape of natural powder or extract from the whole bark.

Therapeutics.—The dominant quality of cascara sagrada is that of producing a positive laxative effect, which may be extended to a violent purging by increasing the doses; from this point of view this drug is analogous to podophyllin.

It may be continued a long time, which permits of its being utilized in habitual and obstinate constipation; and here is indeed its only indication. Nevertheless the American doctors recognize in it a certain action against malarial fever of the remittent or intermittent type.

Mode of Employment.—In the form of powder, the cascara sagrada is taken in doses of from grains vijss to xv in three or four portions or wafers, and this medication, continued several days, will regulate the stools in cases of constipation; to insure the benefit of this effect it is a good practice to continue the administration of the drug at near intervals.

The fluid extract, which responds to the same indications as the powder, is employed in identical doses, that is to say, from grains viiss to xxiiss each day; it may be combined with nux vomica and given in cases of atony of the muscular tone of the stomach and in flatulent dyspepsia.

Because of the violent colics which it may cause, it is somewhat important to refrain from too great increase in the doses of cascara sagrada.

Chloride of Methyl.

Methyl-chlorhydric ether, C^2H^3Cl., discovered in 1834 by Dumas and Péligot. It is a gas of ethereal odor and sweet taste, and its density is 1,736.

Physiological and Therapeutical Effects.—These effects rest upon the fact that the chloride of methyl liquefies at −22 degrees, boils at 36 degrees, and that if after liquefaction a vaporized jet of this ether is thrown upon any body whatsoever the temperature of this body will fall to −22 degrees, and even lower, if evaporation is accelerated by a current of air.

The methyl-chlorhydric ether is a local anæsthetic, a property that it owes to the lowering of temperature undergone by the bodies with which it is brought into contact, and of which its extreme volatility is the cause. First used by Richardson in 1862, it was drawn from oblivion in 1884 by Debove, who utilized it in sciatic neuralgia; then the Drs. Olive, Bucquay, Rendu, Desnos and many others applied it in various neuralgic disturbances, with variable but generally fortunate results.

The first effect which the projection of this gas produces upon the skin is a painful sensation, strongly marked; then, in a few seconds, the skin becomes pale and hardens, and at the same time the pain due to vaporization as well as the original pain disappear. The advantage of this revulsion by the refrigerant action of the chloride of methyl is that, according to Debove and Rouillon, it produces useful modifications in the terminal fibres of the sensitive nerves extending over a large surface, and further it does not injure although it momentarily congeals the integument.

Unfortunately this last proposition is not always confirmed by facts. Besides the phenomena of reaction which ought always to follow so intense a refrigeration, and which decrease within a few days, it is constantly possible for more serious consequences to arise, and they have arisen; in reality it is difficult to graduate the duration and the degree of the cooling in proportion to the resistance of the skin of the subject under treatment; hence possible vesication and escharization.

This said, it must be admitted that the chloride of methyl has proved efficacious.

In unilateral, idiopathic sciatica, sometimes one *séance*, and very often two *séances* have sufficed to effect a cure after a rapid pulverization over the course of the nerve. Debove claims to have succeeded more than a hundred times without producing an eschar. Not a few surgeons report equally numerous successes.

In double sciatica symptomatic of medullary affection, the amelioration is more transient, less rapid and less certain. In neuralgia, intercostal, facial or ilio-lumbar, a few happy results have been noted, as well as in cases of rheumatic pains muscular or articular.

Tonneson has succeeded in overcoming the symptomatic pain in the side dependent upon pleurisy, pneumonia or pulmonary tuberculosis, and without aggravating the internal thoracic lesions.

In that it acts rapidly upon the element of pain, the chloride of methyl has proved on the whole a sovereign remedy for idiopathic neuralgia; it has no influence upon the primitive lesions giving rise to peripheral pains. The difficulty of applying it is the only reason why its employment should be reserved for cases where other means have already failed.

Mode of Administration.—In the application of the chloride of methyl, it is necessary to bear constantly in mind the following precepts laid down by Debove.

Do not direct the jet perpendicularly upon the skin, under penalty of producing a depression which may become the point of departure for a blister or an eschar. Always work upon a large surface, proportion the duration of the application to the thickness of the skin, avoid points where the teguments immediately cover the bones, act with redoubled prudence when dealing with dyscrasic subjects; finally, use only a perfectly pure methyl-chlorhydric ether.

Cocaine.

An alkaloid, extract of erythroxylon coca, which has for formula $C^{34}H^{21}NO^{8}$. It crystallizes in colorless, inodorous prisms of slightly bitter taste. Little soluble in cold water, it dissolves more easily in boiling water and alcohol.

Discovered in 1859 by Niemann, its analgesic properties were observed and occasionally utilized separately, so that we must come to the labors of Coupard and Bordereau, in the laboratory of Dr. Laborde in 1882, to find signalized the abolition of the ocular reflex, insensibility to pricking and pinching, and pupillary dilatation.

From the laboratory, cocaine soon passed into practice, and it was Koller of Vienna who first introduced it into ocular

therapeutics (1884). Since then innumerable observations on the subject of the new medicine have been published, and its domain as a local anesthetic has been considerably extended. Let us add that its feeble solubility has caused preference to be given to its salt, much more soluble than itself, the chlorhydrate of cocaine.

Physiological Effects.—The effects are local or general.

Local Effects.—It is the local action of cocaine upon the ocular mucous membrane that has made its fortune, and the first labors of Koller bear upon this subject. Experimenting upon the rabbit, after the instillation of a few drops, 5 to 100, this author saw the look become fixed, the pupil become dilated, at the same time that the cornea and the conjunctiva remained insensible to touch, to pricking and to cauterization. The experiment renewed upon man produced the same results: first a sensation of dryness at the moment of instillation, then, little by little, the sensibility of the membranes of the eye disappear, the reflexes are abolished, and, even if sensibility to touch is vaguely preserved, analgesia becomes complete. Neverthless the deep parts, such as the iris and the ocular muscles, are painful on section unless care is taken to renew the application of the cocaine as often as these parts are approached. Besides, the action of cocaine is cumulative—that is to say, the anesthetic effect is emphasized and prolonged if care is taken to repeat the doses; an anesthesia of ten minutes is produced by one instillation, and this may be extended to thirty minutes by renewing the application of the collyrium.

The pupillary dilatation caused by cocaine is less important than that produced by atropin, and moreover it hardly continues beyond from eight to ten hours; eserin causes it to disappear rapidly.

Koenigstein has noted, besides, the pallor of the conjunctiva due to the constriction of the vessels, a phenomenon that has been equally observed in the other mucous membranes.

In fact, in the sense of analgesia and anesthesia all the mucous membranes are impressed by cocaine. For a long time Fauvel and Coupard had been utilizing the anesthetic action of coca in affections of the pharynx and larynx; the discovery of cocaine enabled it to be successfully used in the same cases by making the application locally. If the buccal mucous membrane is painted with a solution of 5 to 100, the general

and special sensibilities are diminished, then abolished, at the end of a few minutes. To bring about the same anesthesia in the pharyngo-laryngeal surfaces, a stronger solution is required (from 10 to 20 per cent.). Insensibility is prolonged if the painting is repeated three or four times every two minutes.

The pituitary body is equally impressed by cocaine, as are also the œsophagus, the cavities of the ear, the urethra, the vulva, the vagina, the uterine neck and the anus; these impressions vary in intensity but are always strongest at the point of application. The skin itself has not been found refractory to the action of cocaine; after painting it several successive times with a solution of 5 to 100, I have been able to remove, without acute pain to the patient, a small lipoma from the lower eyelid.

General Effects.—Neuro muscular hyper-excitability and generalized analgesia are, in the opinion of Laborde, the two salient phenomena of the general action of cocaine. These facts have been confirmed by the experiments of Bordereau and Coupard, upon the guinea-pig, of Grasset upon the monkey, of Vulpian upon the frog, and by the clinical researches of Dujardin-Beaumetz, Gougenheim, Morselli, etc.

In subcutaneous injections, cocaine accelerates the respiratory rhythm, which soon becomes irregular; the pulse becomes more frequent, and the blood tension in the vessels is raised; calorification is increased, and this by so much more as the thermometric rise coincides with a greater discharge of heat (Ch. Richet); and, while the other secretions are not modified, salivation becomes more abundant.

The perturbation of the central nervous system constitutes the dominant part of the action of cocaine.

After an injection of $\frac{6}{15}$ of a grain Coupard and Bordereau have in the guinea-pig met with generalized convulsions particularly associated with opisthotonos. Grasset, experimenting upon the monkey, has witnessed convulsive attacks without noting hyperemia. This author has proved the antagonism which exists between chloral and cocaine from a thermic point of view, while antipyrin remains without effect in the same connection. A violent motor impulse has been witnessed in the monkey by Laborde.

The initial excitation is followed by a period of depression,

which varies, according to the dose administered, from simple indolence of the limbs to complete immobility; the reflexes are abolished. Finally, a fatal dose causes stoppage of the heart in diastole.

We must conclude from the foregoing remarks that cocaine ought to be handled with caution. Thourens and Laborde have observed in certain subjects, after a period of initial excitation, a phase of depression with vertigo, pallor of the face, nausea and vomiting.

Dujardin-Beaumetz has four times observed syncope following on injections of cocaine. C. Paul has seen nervous women, under the same influence, brought almost to the point of hysterical attacks. All this ought to cause us to watch carefully over the state of the nervous system after injections of cocaine.

Method of Action.—Of the different opinions put forth by experimenters on this subject, the most general is that which attributes the effects of this substance to a direct action upon the nerve extremities, determining, with insensibility, the contraction of the vessels and consequently a localized anemia; to anemia of the brain might be attributed the nervous disorders consequent upon subcutaneous injections, with so much the more show of reason as these disorders have appeared in subjects who had received the injection standing, and never in those who were lying down at the time of the experiment.

Therapeutics.—In ocular therapeutics, cocaine may be used as an operative anesthetic, or, in painful affections of the eye, as an analgesic.

In the first case, after two or three successive instillations, two minutes apart, with a solution of 5 to 100, the operation for cataract can be performed without the slightest pain. The section of the iris remains painful, but this suffering can be avoided by renewing the application at the moment of the opening of the anterior chamber.

By renewing the instillations as the operation advanced, Abadie has been able to perform during complete anesthesia tenotomy of the muscles of the eye, and Koenigstein goes further in affirming that, following the same method, he has performed without pain enucleation of the eye-ball. In every case, the fact vouched for is complete anesthesia of the cornea and conjunctiva in all operations bearing exclusively upon these

parts. Cocaine has been used as an analgesic in photophobia and acute maladies of the eye; but here its success is only partial, the action of the drug being considerably restricted by the fact of inflammation of the conjunctiva or the cornea.

Affections of the buccal mucous membrane have benefitted by the properties of cocaine, and dentistry has utilized them to soothe the pains of extraction of teeth; amygdalotomy has been performed by Larmayez without suffering on the part of the patient, and I have myself obtained an identical result.

The painting of the pharynx diminishes the reflexes, facilitates laryngoscopic examination and cauterizations of the pharynx or the vestibule or the glottis. The same operation can stop in consumptives the vomitings which follow meals; to this end C. Paul has succeeded with pulverizations of a solution of 1 to 50. Incoercible vomitings and œsophagotomy are amenable to the same means. (Holtz-Domaschino.)

Operations upon the nasal fossa are rendered less painful by preliminary painting with a solution of cocaine of 5 to 100; and cases have been reported where acute coryza was cured by the use of the same solution; this is due to the property mentioned above which cocaine possesses of producing local anemia of the mucous membrane at the point of application.

In affections of the ears, comfort has been obtained from injections of a solution of 1 to 20 for small operations relating to the external auditory duct, and the pains of otitis have been soothed by the introduction of the liquid into the middle ear through the Eustachian tube.

Grynfeldt of Montpellier has succeeded in quieting a painful spasm of the urethra preventing all catheterism by the use of an injection of 2 to 100. Vaginismus and pruritus vulvæ have been soothed or modified by cocaine; Doléris has been able to diminish the pains of parturition by painting the neck when the dilatation had attained the dimensions of a two-franc piece.

Grasset has shown that the subcutaneous injection of cocaine determines an anesthetic zone which permits the performance upon the skin of short and superficial operations, and, by applications of the same drug in solution up to 40 per cent., Hergott has relieved the pain from fissures of the nipple.

Mode of Administration.—We have said that the slight solubility of cocaine has caused preference to be given to its

chlorhydrate, and that it is most conveniently used in the form of solution. This should be in the proportion of 50% for anesthesia of the eye, the vulva, the vagina or the urethra. To act efficaciously upon the olfactory, bucco-pharyngeal or auditory mucous membranes, the solution must be increased to 10 or 20 per cent.

Cocaine may be made into an ointment with oil, lard, vaselin, or lanolin. In case we resort to sub-cutaneous injections of cocaine in order to anesthetize a limited region of the body or to quiet neuralgic pain, we must never lose sight of the accidents which may follow, and we should always begin with doses of $\frac{1}{6}$ to $\frac{1}{3}$ of a grain, which are almost always sufficient.

Finally, we would add that in the exceptional cases where we wish to act directly on the gastric mucous membrane to abate attacks of gastralgia, we may increase the dose of cocaine, which in these cases has been given without risk to the extent of $7\frac{1}{2}$ to 15 grains.

DROSERA.

Drosera rotundifolia, family of the Droseraceæ.

Physiological Effects.—The corrosive properties of drosera have been known since the time of Linnæus, who advised the application of the juice of the plant for the destruction of worms, and, empirically, the same drug was used in catarrhal bronchitis, whooping-cough and influenza. In 1878 Dr. Louvet of Saint-Germain drew the attention of physicians to the action of this plant. According to him, if we order the tincture of drosera in the second stage of whooping-cough, when the cough is very paroxysmal, marked palliation is obtained, and this leads to cure in two to three weeks; in every case complications are prevented. These observations have been confirmed by Beaumetz, C. Paul, Herard, etc. Dr. Lamare adds that drosera manifests its effect on the paroxysms which accompany certain cases of bronchitis and tuberculosis, but here the result is less constant and marked.

The want of success which has followed the use of drosera in laryngitis is explained by the fact that the drug acts exclusively on the spasmodic element and not at all on the anatomo-pathological element. Indeed the method of action of drosera is

not known, for chemical analysis has not revealed any principle which could be deemed armed with special property.

Method of Administration.—Curie has shown by experiment, in particular on himself, that the drosera could be given in large dose as the tincture; he took as much as 3⅓ ounces of the tincture in one day. Lamare determined the same fact, and has given to children of seven 2½ drachms of the tincture daily; he found, however, that these large doses were not more effective than smaller ones (15 to 30 grains).

P. Vigier prefers the following formula to the tincture.

Extr. droseræ, ...	ʒ j gr. xv.
Pulv. glycyrrhiz., ...	ʒ ij gr. xv.

Ft. pil. No. 100.

Four to five of these are to be taken daily.

Duboisin

is an alkaloid obtained from duboisia myoporoides, and was discovered simultaneously by Petit, in France, and by Gerrard, in England. It is isomeric with atropin ($C^{34}H^{25}NO^{6}$), and presents the same reactions, except the brown color which nitric acid yields when added to it.

Physiological Effects.—Brankroft was the first to recognize in duboisia the same properties as atropin; Tweedy in England, and Galezowsky in France confirmed this (1878). The first experiments were made with the extract; the discovery of the alkaloid replaced it.

Locally duboisin does not irritate even when administered sub-cutaneously. Its most important action is on the eye. It quickly induces paralysis of accommodation and mydriasis much better than does atropin, and this remains longer at its maximum, although the total duration is less than with the alkaloid of belladonna. Another superiority of duboisin is that it does not irritate the conjunctiva, no matter how long used.

Experiments on cold-blooded animals have proved that duboisin accelerates the circulation and increases arterial tension, while it diminishes respiratory energy; while in man the latter is increased in proportion to the work of the heart. Quite a large dose causes scarlatiniform redness over the entire surface of the body. The secretions, especially the saliva

and the perspiration are diminished or suppressed as with atropin, even though pilocarpin has been first administered.

The nervous system is also paralyzed by duboisin; Gubler, Fauqué, Galezowski have noted accidents of this nature, especially in tubercular and impressionable subjects.

Therapeusis.—We have stated that the more rapid and complete paralysis of accommodation obtained with duboisin, caused it to be preferred to atropin, a precious advantage for the exact measurement of refraction in myopia and hypermetropia.

Further when conjunctival irritability contra-indicates the continuous use of atropin in ocular diseases, duboisin may be efficiently used in its stead, since it produces the same physiological effects without having the same disadvantage. Rare exceptions invalidate this rule of conduct.

Let us note, however, that duboisin has less effect on inflamed eyes than on these organs when they are in a healthy state, and that as a destroying agent of adhesions it has not proved itself superior to atropin.

In profuse perspiration duboisin may be used as an adjuvant to atropin, and Gubler has reported a case where it possessed properties analogous to digitalis, in a tubercular where the pulse was increased, as also the urinary secretion.

Dujardin-Beaumetz has used duboisin in exophthalmic goitre; at the outset, the redness of the eye, the dyspnea, the precordial pain, diminished, at the same time that the tumor ceased to beat, and that the general state improved. But this remission has never been more than temporary; at the end of a few weeks the palpitations, the exophthalmia and the general bad condition have reappeared; the goitre alone has remained improved.

Desurs, who tested the drug similarly, obtained the same results.

Gubler endeavored to make use of the stupefying action of duboisin on the nervous system, and in a case of alcoholic delirium he obtained the sedation by means of the sub-cutaneous injection of $\frac{1}{85}$ of a grain of the substance which he had vainly sought for through the administration of $\frac{3}{4}$ of a grain of morphine with 45 grains of chloral.

Method of Administration.—The most commonly used collyrium is the following.

Duboisin sulph., ...	gr. $\frac{1}{8}$ to $1\frac{1}{2}$
Aquæ destillat., ...	ʒ iiss.

It may also be used as an ointment, $\frac{3}{4}$ of a grain of duboisin to ʒ iiss of vaselin.

Internally, in case of perspiration, pills containing $\frac{1}{120}$ of a grain may be given once or twice daily. It is always best to begin with one.

It should ever be remembered that the drug is cumulative, and that too frequent dosage might cause collapse and toxic signs similar to those of belladonna.

EUCALYPTUS. EUCALYPTOL.

The eucalptus globulus is a native of Australia, grows quite well in the south of France, especially at Nice; it belongs to the genus Myrtaceæ.

Therapeusis.—Eucalyptus has the reputation of purifying malarial districts, and the inhalation of its odor is said to prevent attacks of fever. It stimulates the organism. The powder is rarely used; the tincture more so, and it answers a number of indications. It may be used internally in bronchial catarrh with fetid expectoration, and then, being eliminated by the respiratory tract, it acts as a disinfectant. It yields also some success in the treatment of uterine catarrh.

Externally it has been used in ozena, but with poor success. In case of sluggish wounds it awakens the vitality of the tissues, and it deserves to be classed among the local excitants of wounds, although its antiseptic properties have been somewhat exaggerated. In our hands a spray of eucalyptus, 1 to 30, has been of good service in tubercular laryngitis.

In a case of confluent variola in a young girl of thirteen, we had the patience to touch each pustule on the face daily, from umbilication to dessication, with the tincture of eucalyptus; the result was that she was discharged without a trace of the disease on her face.

By distillation of the leaves of eucalyptus we obtain an essential oil, eucalyptol. This is a slightly brownish liquid, of a special strong odor, with a burning taste, and which is considered by some chemists to be a mixture of turpentine and cymene.

As a local disinfectant eucalyptol has but little use except

when incorporated into medicinal pessaries. Internally its disagreeable taste renders it non-acceptable to patients, even when administered in capsules. The discovery of liquid vaselin has enabled us to use eucalyptol subcutaneously; shortly the breath of the patient has the characteristic odor of the oil, which is almost entirely eliminated by the pulmonary surface; thus it has been possible to cure fetid bronchitis, and to calm the cough and ameliorate the condition of consumptives.

Eucalyptol has been used as a disinfectant in blenorrhœa.

Method of Use.—The powder of the leaves is ordered in the dose of ʒ j to ℥ ss daily, plain, or mixed with an opiate.

The tincture may be given in the dose of ʒ j to ℥ ss in mucilage.

As for eucalyptol it may be given in capsules to the extent of 15 to 30 grains daily, or subcutaneously in liquid vaselin.

The use of eucalyptol spray in pulmonary affections has given poor results and has caused serious intestinal derangement.

Hamamelis Virginica

Belongs to the family of Saxifrageæ, and grows in the United States, especially in the State of Virginia. No alkaloid has as yet been discovered, but only an essential oil, which, hypothetically, is said to have the medicinal properties of the concentrated plant.

Physiological Effects.—Known and used from time immemorial by the Indians, this plant has latterly been much written about. Hugues and Preston (1874) have noted its value in case of bleeding hemorrhoids, and Hale (1879) considers hamamelis as a hemostatic and sedative, and recommends it in affections of the venous system, against passive hemorrhages and in a certain number of painful affections. Massis records wonderful success from its use in varices. He administered it internally. Most American physicians are similarly enthusiastic.

In France hamamelis has been especially studied by Campardon, D. Beaumetz, Gerrand, Tison.

Guy has shown from experiments on frogs and on rabbits that the plant is not toxic even in high dose. No ill result has ever followed its use.

Hamamelis acts in particular on the venous system, and it has proved especially useful in hemorrhoids, in particular when to the internal administration is added the use of suppositories or of ointments. In a few days the bloody discharge is suppressed and the pains cease.

In varices its action is less certain, and side by side with the success obtained by some we must refer to the want of success in the hands of D. Beaumetz; the disappearance of pain is the phenomenon most frequently noted.

Local congestion is benefited to a degree by hamamelis through the constringent action it has on the vessels; it is thus that at the outset parenchymatous metritis, ovaritis, epididymitis, orchitis, may be aborted.

As a hemostatic hamamelis is not superior to the astringents in ordinary use.

Indeed, the introduction of this substance in French therapeusis created an enthusiasm which has proved quite groundless. Still, hamamelis may render good service in certain cases, such as hemorrhoidal congestion and inflammations in the formation stage.

Method of Administration.—Internally the fluid extract is much used in the dose of a teaspoonful every three hours; this preparation is disagreeable to the taste and must be given in an appropriate vehicle.

Campardon has given the tincture in the dose of five drops, frequently repeated up to 40 drops.

Externally an ointment may be made as follows·

Tr. hamamelis, ...	gr. xlv to ʒ j.
Lard, ...	℥ j.

Suppositories may be made:

Extr. hamamelis, ...	gr. jss.
Butyr. cacaon, ...	ʒ j gr. xv.

A strong decoction may also be made for lotions.

HELENIN.

Helenin is a concrete oil derived from the root of the Inula Helenium. It appears in the form of white crystals, like those of sulphate of quinine, of a bitter taste and aromatic odor; it is insoluble in water, but soluble in alcohol and ether.

Therapeusis.—This medicament, from its first appearance,

has been largely used by Spanish physicians, who claimed that it was a specific against the tubercle bacillus. It causes vomiting in large dose; infants are particularly intolerant of the drug.

In pulmonary affections it acts as an antiseptic and preservative; the first property, as we have stated, has caused it to be used in phthisis; under its influence the cough and expectoration diminish, the appetite and the strength improve; but this amelioration is of short duration, with the exception of the fetor of the expectoration. Helenin, indeed, seems to be rather indicated in bronchorrhea or gangrene of the lung. In the majority of cases this latter affection has been markedly modified by the use of helenin.

The antiseptic properties have been utilized by Korab in infantile diarrhea, and he claims success.

Othiel has applied to diphtheritic membrane a solution of helenin in almond oil, every four hours, and helenin was at the same time given internally. This method failed completely when the disease was five to six days old.

Helenin should be pure, perfectly white, with no resinous odor, and giving no precipitate when added to water or alcohol.

Dose.—The daily dose should not exceed 1½ grains, in divided doses to avoid emesis. A good combination for use in pulmonary affections is the following:

Helenin, ...	gr. vj.
Terpin, ...	ʒ j.
Creasote, ...	gr. ix.

Div. in pil. No. 25.

Four to five of the above pills may be taken daily.

Hypnone.

Hypnone (*acetophenone, phenymethylacetone*) was discovered in 1857 by Friedel, and has been studied from a therapeutic point of view latterly by Dujardin-Beaumetz and his school.

Physiological Action.—From experiments on animals Drs. Mairet and Combemale, of Montpellier, have been able to state the following deductions:

The immediate effects of hypnone on animals, aside from slight transient somnolence, leaving the senses awake, have nothing in common with sleep. Acetophone is only slightly

toxic, but there are consecutive effects which should make us cautious in its use. When a large dose has been given an animal, we note a loss of weight and of hemoglobin; the same happens to a dog when he ingests daily small doses of acetophone.

In man the effects differ:

In from twenty minutes to one hour hypnone causes sleep, calm, deep, and he awakens readily without nausea or distaste for food, as happens when he has taken chloral and paraldehyde; sometimes there exists supra-orbital cephalalgia.

Some patients are not at all affected.

Hypnone has no analgesic effect, but increases that of chloroform. It seems to diminish nervous excitability; it depresses sanguineous pressure, an indispensable condition for the production of sleep.

Hypnone has no action on the stomach, aside from occasional heat or nausea when, in particular, hypnone is associated with ether in capsules.

On the blood hypnone produces the following effects, according to the experiments of Quinquaud and Laborde: an injection into the circulation of 30 to 45 minims of hypnone produces, in the dog, an increase in the carbonic acid of the arterial blood; the oxygen is further lessened in amount, the phenomena of asphyxia are therefore obtained when the dose is sufficient. The glycogenic function of the blood is doubled.

All the animals used by these experimenters died, and this should teach us to be prudent in the use of this remedy.

Therapeusis.—The hypnotic action of acetophenone might be utilized in the nervous insomnia following on alcoholic excess or intellectual labor. In these cases hypnone is superior to other agents provided the patient has not been subjected to the treatment by morphine. A febrile state contra-indicates the use of hypnone.

Method of Administration.—The preferable method is in perles or capsules containing ¾ of a grain.

The maximum dose is 7½ grains, but we must give at once 4½ to 6 grains in order to induce sleep.

ICHTYOL.

The introduction of this new remedy into therapeusis is due to Unna of Hamburg. Schrœtter discovered it by distillation

from a bituminous substance found in 1881 at Seefreld in the Tyrol. This bitumen is none other than the decomposed residue of animal matters coming from fish and pre-historic animals. This hypothesis is based on the presence of a large number of fossils, and imprints of fishes, in the layers of the bituminous rock; whence the name ichtyol.

It occurs in the form of a blackish mass of the consistency of vaselin or of thick oil. Its odor is strong and disagreeable; its chemical composition is ill-defined, but it contains sulphur and phosphorus in notable quantity.

Ichtyol forms an emulsion with water, but is soluble in part in ether and in alcohol; a mixture of these two dissolves it rapidly; it mingles readily with vaselin and oils.

Therapeusis.—Ichtyol has been used in diseases of the skin and in certain internal affections.

Unna first used it in case of an inveterate psoriasis, without effect; next in eczema and in thirty consecutive cases he obtained wonderful results. Among these cases were some of moist eczema of the arm and the hand, a case of papular eczema with intense pruritus, a number of cases of ambulant eczema of the lower limb, two cases of humid eczema of the head in infants, and an extensive eczema in an infant of three months.

The medicament was used as a 5 per cent. ointment, gradually using as high a percentage as 100, and he noted the rapidity with which the ichtyol dried up the eczema and quieted the pain and the itching. In some cases he used a spray of ichtyol in a mixture of ether and of alcohol.

Unna remarks that the effects depend on the dose of the substance, and advises the use of gradually increased strengths.

The state of the epidermis will serve as a guide; the more healthy this is the stronger should be the ointment; thus for a dry eczema we may use a 50 per cent. ointment, while for a moist a 20 per cent. suffices.

The most marked effect of ichtyol is to quiet the pain and pruritus.

This substance is not at all incompatible with lead or mercury, and in some cases these may to advantage be combined.

The formula recommended by Unna is:

Litharge, ...	ʒ iiss.
Vinegar, ...	℥ j.

Boil down to one-third and add:

Olive oil,	
Lard,	āā ʒ iiss.
Ichtyol,	

Ichtyol has proved useful in the hands of many dermatologists in acne, favus, prurigo.

Rheumatism has in some cases been benefitted by ichtyol; acute rheumatism, of mean intensity, treated by applications of a 10 per cent. ointment, the joints being wrapped in cotton, has been as quickly cured as by the use of salicylic acid.

Chronic rheumatism requires a stronger ointment: 2 to 50 per cent. In the torpid forms pure ichtyol may be used, washing the skin with soap and water beforehand.

The following solution in lotion or in spray has given good results:

Ichtyol, ...	ʒ iiss.
Castor oil, ...	ʒ v.
Alcohol, ...	℥ iij ʒ iiss.

In certain cases the pains have been greatly relieved by inhalations of ichtyol, and Unna has resorted to this method with success in influenza and in bronchial catarrh with fever and generalized pains. A dessertspoonful of ichtyol is added to 1 to 2 quarts of water, this is gently heated and the fumes are inhaled.

This method has clearly been successful in case of recent coryza with abundant secretion, and in catarrh of the larynx and trachea accompanied by abundant hypersecretion.

A beginning coryza is often aborted by a ten minutes' inhalation.

In angina, at the outset, the following spray every hour has given good results:

Ichtyol, ...	ʒ j gr. xv.
Ether. sulphuric.,	
Alcohol	āā ℥ j ʒ v.

Externally ichtyol has the disadvantage of producing a miliary eruption; this may be prevented by not covering the parts with objects which provoke perspiration, such as cotton, or by mixing with the solution of ichtyol a preparation of lime, as in the following formula:

Ichtyol, ...	ʒ iiss
Olive oil,.	
Lime water, ...	āā ℥ iij ʒ iiss.

An alcoholic solution of cumarin and of vanilin will suppress the disagreeable odor of the ichtyol.

Iodol.

Iodol, or tetraiodopyrrol, was discovered in 1883 by Silber and Ciamician; its formula is $C^4/_4NH$. It is a slightly brownish powder, amorphous, almost without taste, and of an odor a trifle like the essential oil of thyme. Exposed to the light its color deepens. It is almost insoluble in water, slightly soluble in alcohol, soluble in ether and in alkaline solutions.

Therapeusis.—Iodol is an antiseptic to the same degree as iodoform; it has been deemed more powerful than the latter, but this is not proven; its only proved advantage is the absence of disagreeable odor and of not causing toxic symptoms.

It is indicated as a dressing for wounds; it acts like iodoform by the slow freedom of small quantities of iodine.

Method of Use.—It is employed in the crystalline form. We recommend, in particular, the following formula:

Iodol,	
Calomel, ...	āā gr. xlv.
Phenic acid (crystals), ...	gr. viiss.

In alcoholic solution it may be prepared as follows:

Iodol, ...	gr. xlv.
Alcohol, ...	℥ j ʒ iiss.
Glycerin, ...	ʒ j gr. xv.

A gauze may be prepared by dipping it in:

Iodol,	
Resin,	
Glycerin, ...	āā gr. xv to xxx.
Alcohol, ...	ʒ iiss.

Jequirity.

Jequirity is the common name given by the Brazilians to the Abrus precatorius, which they have used for long in ophthalmia, and which de Wecker introduced into France.

In 1882 this gentleman reported to the Academy the results of his experience with washing the eyes with a maceration of the grains of jequirity. A purulent ophthalmia resulted which, in resolving, cured conjunctival granulations.

A little later Moura-Brazil claimed priority in the use of jequirity.

De Wecker's results caused ophthalmologists to use the drug with enthusiasm. In France, in Belgium, in Germany, numerous cases were reported, but unsuccessful results rather dampened the ardor with which jequirity was first received.

Method of Use.—The grains of jequirity are macerated for twenty-four hours in cold water, in the proportion of 2 to 5 per cent., this is filtered and used at once, the internal surface of the lids being washed and the conjunctival culs-de-sac being irrigated.

A cold maceration is preferable to a hot, and the liquid must be used at once. Antiseptic substances should not be added.

Five per cent. solutions should be infrequently used, at the end of twenty-four to thirty-six hours, else corneal accidents may occur, and such have been reported.

Physiological Effects.—De Wecker says: "The use of such solutions causes a croupous inflammation, intense according to the number of applications and the strength of the solution. The cornea runs absolutely no risk during the evolution of the ophthalmia."

The immediate pain is *nil;* this appears only at the end of three hours. Hyperemia first appears, and next swelling; a purulent fluid pours from the eye, and at the end of twenty-four hours the artificial ophthalmia attains its maximum. A species of whitish false membrane covers the conjunctiva of the lids, and if it is removed a second forms on the following day. Underneath it the conjunctiva is swollen and bleeding. Chemosis may result if the solution was too strong, and then, the opinion of de Wecker to the contrary, the cornea is in danger. Such a complication is unlikely with weak solutions, the reaction being always proportionate to the concentration of the liquid.

The inflammation provoked is acute in proportion to the severity of the primary conjunctival affection, and the use of a pencil of jequirity increases the acuity.

At the end of a week the resolution of the jequirital inflammation sets in; the symptoms improve of themselves, and, at the end of two to three weeks, the process is at an end.

The general symptoms are but little marked; a little fever, cephalalgia; and, at times, vomiting.

Let me add that the conjunctiva is not alone influenced by jequirity. Other mucous surfaces present the same reaction, also the skin to a lesser degree.

Method of Action.—Microscopic examinations added to physiological experiment have shown that the special action of jequirity is due to a microbe described by Sattler in 1883. On culture this microbe gave birth to products which, applied to the conjunctiva of animals, have reproduced an exact jequirital ophthalmia. Boiling, further, destroys the properties of the seeds of *abrus precatorius.*

The experiments of Cornil and Berlioz confirm this theory; these writers, on injecting a few drops under the skin of chickens, observed the same phenomena as those of cholera; further the local reaction was very great. Introduced into the peritoneal cavity a croupous inflammation followed. The injection of more than ten drops caused death.

These experimenters often noted in animals what has been seen in man: a first inoculation protects.

None of the above phenomena were noted when the maceration had been sterilized.

Notwithstanding these conclusive experiments, Bruglants, Schmidt, and, above all, Deneffe, see only a chemical action, but Martineau has completely refuted this view by introducing a strong solution of jequirity into the vaginæ of women and never seeing inflammation follow as long as a current of air was directed into these channels, a fact which dispels the chemical theory.

Therapeutic Application.—The capital indication for jequirity is granular ophthalmia; this medicament has an unquestionable value, although many deny this.

Wecker says:

"Jequirital ophthalmia certainly quickly cures granulations, and acts with far greater safety than blenorrhagic inoculation.

"After a rational use the conjunctiva does not become absolutely healthy; but it is uniformly covered by a smooth epithe-

lium, and no further treatment is needed to hasten the disappearance of the corneal lesions."

Jequirity succeeds the best in the dry, chronic form of granular ophthalmia, which is accompanied by inveterate pannus; it is, indeed, the best agent at our disposal against conjunctival and corneal trachoma. The same does not hold true of slight granular conjunctivitis, where the sulphate of copper and acetate of lead act better.

Jequirity is contra-indicated by the presence of purulent ophthalmia, whatever its nature, and blenorrheal inoculation of an eye which has been subjected to jequirity brings about deplorable complications.

In justice we must cite the opinions of those who have not been able to endorse jequirity. Ferrier has not had good personal results; Galeznoki claims that after a brief amelioration the disease recurs as bad as ever; Gayet does not praise jequirity, and Deneffe rejects it as absolutely inefficacious.

Among other applications of jequirity we would note that Tangenaur has used it in median purulent otitis; after injection into the external auditory meatus, he has seen a violent inflammation develop, and at the end of this inflammation the otitis was cured. The results were especially remarkable in case of fungosities of the middle ear.

Shoemaker proposed the use of jequirity in neoplasms of the skin, lupus, epithelioma, papilloma, etc. He applied a concentrated solution; at the end of a few hours a crust forms which is shed under poultices; below cicatrization is going on. He reports a number of successful cases. He advises care in the use of the agent, since it might induce local or general complications of greater or less degree.

Kairin.

Discovered by Otto Fischer (Munich) in 1882, kairin is nothing else than the chlorhydrate of an alkaloid, the chemical name of which is oxyhydromethylquinolin ($C^{20}H^{13}NO^2$). It is a fine powder, clear yellow, musk-like in odor, aromatic and bitter in taste, insoluble in ether, very soluble in water and in alcohol; these latter sometimes change quickly on exposure to air. Filehne of Erlangen first used it on account of its powerful antipyretic properties, and it was next studied by

Hallopeau, Broudardel, Girat, and others in France, and abroad by Merkel, Renzy, Morokowitz and others, who, while admitting its value have also exposed its disadvantages.

Physiological Effects.—Kairin acts on all the functions of the economy, and it will be useful to pass this individual influence in review:

Calorification.—In warm-blooded animals kairin determines a rapid but transient fall of temperature. Girat and Conscience have noted that in health very large doses were requisite to obtain a fall of one degree, and that toxic doses were requisite for a greater fall; exception must be made in favor of the dog, in which a fall of 4.6° to 9° was obtained. The beginning of the fall of temperature has always been marked by violent chills, which reappeared as the temperature rose again; further, the renal congestion evident on autopsy and the presence of blood in the urine proved that kairin in high dose is irritant to the eliminators. Filehne had already noted that man in a state of health was little or not at all influenced as regards temperature by small doses of kairin, contrary to what happens in fever where the fall is constant.

Circulation.—With rare exceptions the pulse is modified, as is the temperature. Arterial tension is constantly diminished, either at the outset or after a slight increase, and at autopsy of animals killed by toxic doses the heart has been found full of clots in diastole.

State of the Blood.—Kairin is an energetic deoxidizer of the blood; it causes a diminution of the proportion of oxygen to carbonic acid.

The blood of animals killed by kairin is black and readily coagulates; a current of air and even of oxygen is powerless to restore the red color; the corpuscles are also modified in color and in form. Spectrum analysis proves that these changes are due to the alteration of hemoglobin into methemoglobin.

Respiration.—The respiratory rhythm about follows the fluctuations of the thermometer; it becomes short, broken, painful.

Digestive Apparatus.—These troubles here are not marked, but we must note the very bitter taste of kairin, the hot sensation to the tongue, and the bluish tint which rapidly spreads over the buccal mucous membrane, and which may extend to the integument—indices of interference with oxygenation.

Nervous System.—After subcutaneous injections, convulsions, epileptiform movements, contractures occur, to which succeed paresis or even complete paralysis. Locally there may be anesthesia in the injected region, and this may extend to other parts of the body if the dose of kairin be high.

All the other secretions are increased.

Rengi, and later Conscience, have proved that kairin has marked anti-putrid properties.

Therapeusis.—Kairin is a powerful antipyretic, but its action is transitory. A dose of 4½ to 7½ grains causes a fall of temperature in half an hour, and the fall attains half a degree and more if this dose be repeated every hour and a half; at the third to fourth hour the temperature has returned to the normal; if the drug is discontinued at the end of two to three hours, the temperature returns to its initial point, and it is necessary in order to maintain the antipyresis to give the kairin at more frequent intervals, although in less dose.

What we have stated applies only to the febrile patient.

In weak patients smaller doses should be used (1½ to 3 grains), and give these doses from hour to hour.

From the start defervescence is accompanied by profuse perspiration, which lasts as long as the temperature is down, and which certainly depresses the strength of the patient.

During the apyrexia the patient feels well, but as the temperature rises, he has a violent chill which shakes the whole body and ends only when the temperature has returned to the point where it was before the administration of the drug, and this lasts about half an hour.

Collapse with cyanosis and emesis have happened but infrequently, but we must remember the likelihood of such an occurrence and always begin with moderate doses.

The dryness of the throat, the pruritus of the nasal fossæ, a little frontal headache, are only accessory phenomena which do not amount to anything.

P. Decaye thus judges kairin: "It determines an apyrexia as certain as it is transient; it always provokes abundant perspiration and a violent chill at the start and at the end; it may cause grave collapse, and this may happen at any time and from any dose, so that the physician or a trained nurse should ever be present."

We must conclude that kairin, having no influence on the

morbid process, should be reserved for cases where the great elevation of temperature constitutes a pressing danger.

It was in typhoid that kairin was first used, and this was a great mistake in view of the powerful means we already had at our disposal, and in view of the formal contra-indication to any agent the first effect of which is the alteration or the destruction of the red corpuscles, when by the presence of the disease itself the state of the blood is profoundly altered.

The same views hold for pulmonary tuberculosis, an eminently destructive process; there also is collapse and dyspnea especially to be guarded against, symptoms which kairin may cause.

On the other hand, pneumonia, according to Hallopeau, Drasche, and others, calls for kairin when the temperature is very high, the pulse is rapid and the dyspnea becomes painful. The drug secures a state of well-being for such a patient even though it be transient.

The same views may be held for subacute rheumatism, for the eruptive fevers, erysipelas, and for all affections where the rise of temperature constitutes an immediate danger and considerable anguish. It goes without saying that paludal fevers nowise call for kairin.

Method of Administration.—The preferable way of giving kairin is by the mouth. It may be given in cachets. High doses, we have stated, should be prescribed, and that the dose should be guided by the thermometric range; 4½ to 7½ grains may be given at once to an adult, and 1½ to 3 to infants or to weakened patients.

Subcutaneous injections are more rapid in effect and more durable, but we must remember the possible local consequences, abscesses having frequently formed in animals, and for this reason this method of administration has not been used in man.

Kola.

Under the name of kola, *Café du Soudan*, we understand seeds which Rabuteau, Heckel, Dujardin-Beaumetz, Monet, have found to contain caffein and theobromin associated with tannin, and fats, and it constitutes a medicament which regulates the circulation, is tonic and anti-diarrhœic.

The true kola is furnished by several varieties of the family

of Sterculiaceæ; the seeds are of variable size, red or white, oblong in shape. Caffein is present in greater quantity than in the best coffee, and is not combined with an organic acid.

Physiological Effects.—The richness of kola in caffein leads us to suppose that it ought to act like this alkaloid, and so it does.

Kola causes insomnia, at least in impressionable subjects; it stimulates the intellectual faculties, and nourishes the organism.

Its influence on the circulation is considerable; it increases vascular tension, slows the cardiac rhythm (after an excitant stage noted by some) and renders the heart beats more regular and ample; this action is even more marked than that of caffein.

Monet believes that kola is a muscular poison, acting directly on the muscular fibre, and in toxic dose that it induces tetanus and loss of muscular excitability. In moderate dose, kola excites not only the heart but smooth muscular fibres, and to this instead of to a truly diuretic influence is due the frequent desire to micturate which follows its administration.

Therapeusis.—Kola, owing to its analogy with caffein, has been used in heart disease; it is useless to reproduce here the advantages of the latter to cardiacs; the advantage of the former, its advocates say, is that it exerts not alone a general tonic action but also furnishes nutrient force to the organism.

Kola is hence better indicated in phthisical or adynamic states, or in convalescence from protracted fevers.

Kola also assists digestion, suppresses vomiting, and ameliorates dyspnea.

Certain physicians (Curieo, Durian) have found kola useful in diarrhea and in cholera morbus; but this view is hardly tenable; kola must be considered as a cardiac tonic, as a general stimulant, as a digestive regulator, as an anti-diarrhœic.

Method of Use.—The preparations of kola are:

Alcoholic extract in the dose of $\frac{1}{6}$ to $\frac{3}{4}$ of a grain.

The tincture, dose of 30 to 150 grains.

The fluid extract, dose of ʒ j ♏xv to ʒ v.

The wine, which is to be preferred where the entire organism needs stimulation.

LANOLIN.

Lanolin is a fat extracted from sheep suet, and it is daily acquiring an important place as an excipient of use in diseases of the skin.

When pure, it is a viscous, glutinous fat, of yellow color, almost without odor and of neutral reaction.

Properties.—The principal property of this body is the rapidity with which it is absorbed by the skin. After friction with lanolin the skin is firmer, turgescent, and yet the surface is nearly dry; this rapid absorption is apparently due to its intimate relation with the epithelial fat.

Another advantage of lanolin is to readily absorb its volume of water, and a very large proportion of glycerin, giving a homogeneous stable mixture, a precious property for the administration of drugs by the skin; the addition of water, indeed, to a fat appears to increase greatly the absorbent power of the skin for this fat and medicinal substances; and while 100 parts of vaselin absorb 4 of water, 100 parts of lanolin absorb 500 of water.

Lanolin does not irritate the skin nor does it become rancid. It mixes readily with metallic mercury and a trituration of a half hour causes the disappearance of all globule even under a magnifying glass.

Concentrated saline solutions are absorbed by this body to the same degree as is water and glycerin.

The rapidity of absorption by the skin of agents incorporated with lanolin has been established by Liebreich, Fraenkel, Kœbeuer, Lassar, and others, such agents being mercury, sublimate, iodoform, iodide of potass, etc.

The only disadvantage of lanolin is the hardness of the ointment, but this is readily remedied by the addition of a little glycerin or lard; by mixture with the latter the absorptive power of lanolin may be readily altered.

We give certain of the formulas used in Berlin, where lanolin was first experimented with:

Extract of Conium Ointment.

Extr. conium, ...	ʒ j gr. xv.
Lanolin, ...	℥ j ʒ iij gr. xv.

Iodide of Potass Ointment.

Iodide of potass,	
Distilled water,	
Lard, ...	āā ʒ iiss.
Lanolin, ...	℥ ii ʒ iss.

Iodoform Ointment.

Iodoform,	
Lard, ...	āā ʒ iiss.
Lanolin, ...	℥ iiss.

Boracic Acid Ointment.

Boracic acid, ...	ʒ iiss.
Lard, ...	ʒ v.
Lanolin, ...	℥ ij ʒ iss.

Phenic Acid Ointment.

Phenic acid, ...	ʒ j gr. xv.
Lard, ...	ʒ iiss.
Lanolin, ...	℥ ij ʒ viss.

Naphthol Ointment.

Naphthol, ...	ʒ j gr. xv to ʒ iiss.
Lard, ...	ʒ iiss.
Lanolin, ...	℥ ij ʒ v gr. xv.

Lead and Phenic Acid Ointment.

Acid. phenic., ...	gr. xxx.
Lanolin,	
Ointment of carbonate of lead, . .	āā ℥ j ʒ ij
Olive oil, ...	ʒ v.
Essence of lavender, ...	gtt. xxx.

Ointment of Tannin and Iodoform.

Tannin, ...	ʒ j gr. xv.
Iodoform, ...	gr. xxx.
Lard, ...	gr. xlv.
Lanolin, ...	℥ j.

In preparing ointments of lanolin the druggist should be directed to gently heat the substance without melting it.

CONVALLARIA MAIALIS.

This agent has been long used and of later years has been resurrected. In the last century water distilled from convallaria was lauded in palpitation of the heart and to stimulate the circulation. Knowledge of this drug, according to some, dates back to the sixteenth century.

In 1880, Troitzky and Bojojawlensky, in Russia, made a series of experiments which proved the powerful influence of convallaria on the circulation. Professor Sée published his experience in France, and since works on convallaria have appeared in all the countries of Europe.

Convallaria maialis belongs to the family Liliaceæ; it contains two alkaloids: convallarin and convallamarin, which represent the toxic element of the plant and also the active element on the circulation; it is an amorphous, bitter powder, soluble in water.

Physiological Effects.—It has long been known that convallaria in high dose was toxic, and the following are the symptoms of poisoning in the dog, according to Sée.

During the first stage we notice slowing of the heart's action and increase in its force; at the same time the respiration becomes a little less frequent, while more ample.

In the second stage the heart presents great irregularities in its contractions, being intermittent with abrupt systole—in a word it acts by jerks, so to speak; the respiratory rhythm also loses its regularity, becoming at the same time slower; the inspiration becomes longer; the respiratory act takes place by a series of convulsions of the inspiratory muscles, as it were.

In the third stage the blood pressure is lowered, the respiratory movements are slackened, the pulse becomes rapid and imperceptible, and death occurs from heart failure. The muscles have retained to the end their contractility and reflex excitability.

As is apparent we have here the picture of poisoning by digitalin; the sole difference consists in the dangerous period which is manifested by the latter, and which is characterized by a prolonged arrest of the heart between two series of contractions regularly slackened.

Extract of convallaria holds the same relation to convallamarin that digitalis does to its alkaloid.

Therapeutic Effects.—It is unnecessary to recall the deleterious action of the flowers of convallaria in a closed room, nor the sternutatory property of the powdered dried leaves; we will only consider convallaria from the stand-point of its influence on the central circulation, comparing it with digitalis.

The first advantage of the extract of convallaria over the preparations of digitalis is that it is well tolerated by the stomach; no nausea, no bitter taste, no vomiting is caused by it; only an increase of the salivary secretion.

Extract of convallaria acts energetically on the heart. When this organ is arhythmic in action from purely functional irregularities without organic lesion, it has restored to it its normal rhythm by this drug. The results are less constant when the disturbances are dependent on a valvular lesion or on hypertrophy.

G. Sée notes with truth the superiority of convallaria over digitalis in the treatment of cardiac palpitations; the first slackens the beats of the heart without checking the excitability of the pneumogastric, while the second energetically stimulates the cardiac muscle if long continued.

The arterial circulation is also favorably modified, and the sanguineous pressure is increased in the vessels, proof of the stimulant property of convallaria.

The respiration becoming more ample and easier procures for the patients a sensation of well-being, which is increased readily by the addition of iodine to the preparations of convallaria.

The drug causes diuresis, which persists for five to six days after its administration, and the quantity alone of the urine is increased without modification in the chemical composition. Exceptionally convallaria has seemed to have an unfavorable influence on the kidneys, and the greater part of the diuretic action must be considered due to the increase in arterial tension.

To resume: Regularization of the movements of the heart and increase in its contractile power and in the blood pressure, increase in the amplitude of the respiratory movements, energetic diuresis, absence of nervous and of digestive disturbances as well as of cumulative effects; such is the physiological

action of convallaria, and therefore Professor Sée recommends it in:

Essential palpitations; arhythmia without organic lesion; uncompensated mitral stenosis; mitral insufficiency complicated with venous congestion; aortic insufficiency; cardiac dilatation, even, accompanied by degeneration of the heart.

All affections of the heart which have produced edema; this latter indication is especially important in view of the powerful diuretic action of convallaria.

The successes obtained by Sée have not been noted by all clinicians, and, without mentioning the opposition in Germany, we must refer to Professor Peter, who claims that the drug is only a good diuretic. Still, Russian and American physicians praise the drug in dyspnea and in dropsy, and recommend it where digitalis is badly borne by the stomach.

Method of Administration.—The aqueous extract of the entire plant is the best preparation of convallaria; it may be given in 15 to 30 grain doses either in pill or in syrup.

Convallamarin should be given in doses varying from $\frac{1}{12}$ to $6\frac{1}{13}$ of a grain; but its action is not greater than that of the aqueous extract.

NAPHTHOL.

Under the influence of sulphuric acid naphthalin breaks up into two acids, one of which has latterly proved of value therapeutically.

Therapeusis.—Used exclusively externally, naphthol has the advantage over naphthalin of being almost odorless and less dangerous to use.

In 1881 Kaposi (Vienna) used naphthol in psoriasis, eczema, prurigo, and soon after Hardy and Besmer, in France, began to employ it.

Rubbed on the sound skin it softens the epidermis, and at the end of a few days it causes slight desquamation; on the diseased skin it causes a scab which lasts only about one half hour.

Naphthol is eliminated by the urine, which turns violet and at the end of a few hours olive green. The elimination as with pyrogallic acid is intermittent.

Kaposi, and after him Jarisch and Neisser, have observed

hematuria and signs of violent renal irritation from repeated friction with naphthol, and they therefore recommend its use, at the outset, in small dose especially in infants and those with delicate skin.

The general opinion of dermatologists is that naphthol is efficient in case of ptiriasis and prurigo, and Hardy affirms that this substance quickly destroys and with certainty parasites, while it quiets the itching; it has the further advantage of being inodorous and of not soiling the linen.

In regard to other dermatoses there is no agreement; while Kaposi reports much success in eczema, psoriasis, etc., Besnier and Sombert declare that it is less efficacious.

To resume: The parasiticide action of naphthol is the sole one established.

Method of Administration.—Kaposi uses an ointment for friction, and then covers the parts with pulv. amyli. The following is the formula:

Naphthol, ...	ʒ iij gr. xlv.
Green soap, ...	ʒ ijss.
Lard, ...	℥ iij gr. xx.

Hardy uses:

Naphthol, ...	ʒ ijss
Vaselin, ...	℥ iij gr. x.

In psoriasis Hardy uses a 15 per cent. ointment, and in prurigo daily inunction with 5 per cent. ointment.

Nitrite of Amyl.

Nitrite of amyl is obtained by passing a current of nitrous gas through the hydrate of amyl heated over a water bath.

Therapeusis.—The inhalation of the vapor of amyl nitrite strongly and quickly congests the brain; the face becomes turgescent and there follows a certain amount of headache. At the same time the heart beats are accelerated, while the peripheral vascular tension is lowered.

The properties of this drug have been used to combat the cerebral anemia which is due to cardiac troubles and to migraine; but it is, in particular, angina pectoris, so rebellious to all methods of treatment, which has been subjected to it.

Sée, Potain, Jaccoud, Dujardin-Beaumetz, have eulogized it, and in many cases they have secured cessation of the suffocative attacks and the disappearance of the intense pain. It suffices to pour a few drops on a handkerchief and to inhale; on account of its intense action it must be used cautiously. In order to render its use easier it has been enclosed in glass pearls, and these may be broken in a handkerchief.

In apoplectic patients the nitrite of amyl is formally contra-indicated.

Nitro-Glycerin.

This body has the formula $C^6H^5O^6 (2NO^4)^3$ and is derived from glycerin by the addition of hypo-nitric acid. It is an oily liquid, yellow, of a burning taste, little soluble in water and in common alcohol.

Originally used in America and in England, particularly by the homeopaths, it has latterly been carefully studied in France by Huchard and Dujardin-Beaumetz, and Marieux has treated of it in a very complete work.

Physiological Effects.—These effects are many, and it is necessary to examine them from several stand-points:

On the circulation nitro-glycerin, in animals, provokes, at the outset, an acceleration of the cardiac contractions, followed by slowing and irregularity; the tension is lowered in the arteries and the peripheral circulation is slackened, owing to the vascular dilatation. In man the lowering of the arterial pressure and the dilatation of the peripheral vessels provokes heat of the skin, which is covered with perspiration; the eyes are injected, there is ringing in the ears, the arteries of the neck beat visibly, and ophthalmoscopic examination proves that the same changes occur in the encephalic circulation; cephalalgia is quite pronounced.

This stimulation of the encephalic and cardiac circulation is the reason for its value in angina and in aortic affections.

On the nervous system nitro-glycerin exercises an action which is manifested by cephalalgia, vertigo and visual disturbances, among which has been noted a transient amblyopia. At the end of a few hours these phenomena are replaced by a general lassitude and desire for sleep. On awakening the organism is in its customary state.

On the respiration nitro-gylcerin has a less marked action;

sometimes the respiratory rhythm is not affected, and this is the rule; when the reverse occurs, increase in frequency and in amplitude are noted.

We have still to refer to the nausea and vomiting which occur in certain patients.

Therapeusis.—The vaso-dilating action of nitro-glycerin is the sole indication for its use, and this agent, far from being a panacea for all the affections in which it has been said to be of utility, should be reserved for cases where ischemia of the cardiac muscle and of the brain calls for it.

Angina pectoris is in the front rank; the general opinion is that this affection is due to cardiac anemia, and that spasm of the coronary arteries is the source of the intense pain. Murrel, Huchard, Dujardin-Beaumetz have obtained favorable results from nitro-glycerin in angina, alone or associated with nitrite of amyl, the action of which is more rapid but more transient.

The affections of the aorta, which cause great thoracic pain, as well as dynamic cerebral troubles resulting from the anemia of the brain, call for nitro-glycerin. It is useful in the vertigo, pallor, syncope which we especially observe in aortic insufficiency.

In fatty degeneration of the heart nitro-glycerin also seems indicated, but here it is questionable if digitalis should not be preferred on account of its tonic action on the cardiac muscle, while trinitrin acts only by diminishing the work of the heart.

The diseases of the nervous system which call for nitro-glycerin are few enough in number; non-congestive cephalalgia, dental neuralgia, angiotonic migraine; the drug, however, is absolutely contra-indicated in the presence of cerebral congestion, active or passive; cephalalgia in the anemic or chlorotic is usefully met by nitro-glycerin.

As for the utility of the drug in the major neuroses it is doubtful and must be further studied.

In chronic nephritis and in pulmonary affections satisfactory results have been obtained, but such has not been the experience of French observers, and we should accept these conclusions with great reserve.

Method of Administration.—Nitro-glycerin may be given by the stomach or subcutaneously.

Huchard's formula is:

Alcoholic sol. of nitro-glycerin, (1:100)	gtt. xxx.
Distilled water, ...	℥ ix ʒ iij.

Of this one to three spoonsful may be taken daily.

Hypodermically the following formula is useful:

Alcoholic sol. of nitro-glycerin, (1:100)	gtt. xxx.
Distilled water, ...	ʒ ijss.

This contains three drops of the solution to each 15 minims.

We should always begin with one to two drops daily to test the susceptibility of the patient, and gradually increase to five drops, which limit should never be exceeded.

PAPAYOTIN.

From the fruits of Carica papaya is obtained a thick, white, milk-like fluid, of neutral reaction, which ferments easily with the odor of butyric acid, and this has been found to have digestive, drastic and vermifuge properties. The digestive property is due to a principle acting like a ferment to which Würtz has accorded the name papaine; it is an amorphous powder, soluble in water, not affected by hydrochloric or nitric acid; in composition it approaches the albuminoids.

Würtz thus explains the action of papaine: it dissolves rapidly large quantities of fibrin, even as does pepsin, but with the difference that solution with the former not only occurs in the presence of a little acid, but also in a neutral or slightly alkaline medium.

Bouchat and C. Paul have proven that papaine acts on fibrin not only as a solvent, but that it also transforms it into an assimilable peptone.

The juice of the papaya is irritating; applied to the raw skin or injected under it, it causes much pain, giving rise to a large abscess; given in large dose by the mouth it purges violently, and it may even cause ulceration of the intestine, a fact proved by Moncorvo in animals.

In Europe only the digestive properties of papaya have been used; and papaine is the only preparation resorted to; it has replaced pepsin in the treatment of chronic affections of the digestive tract, for the latter substance is often inefficacious on account of the manner in which it is prepared.

Method of Use.—Papaine is given in the dose of 1½ grains in a spoonful of syrup at meal time, or else in cachets. It should not be given in alcohol, as this renders it insoluble.

Bouchat has proposed obtaining the cure of certain solid tumors by the interstitial injection of papaine, and Péan has used such injections in three cases of cancer of the breast, and he has obtained digestion, for the aspirated fluid seemed to be a true peptone; the injection, however, is very painful and has always provoked much fever.

PARALDEHYDE.

Paraldehyde ($C^{12}H^{12}O^{6}$) is a liquid of a strong and quite agreeable odor, of a burning, acrid taste, colorless, soluble to a greater degree in cold than in hot water.

Cervello introduced it in 1882, having discovered its hypnotic properties and its antagonism to strychnine.

Physiological Effects.—Dujardin-Beaumetz and Coudray subjected frogs, guinea-pigs and dogs to the action of paraldehyde. Their results may be stated as follows:

Sleep was always obtained, and when administered hypodermically, local paralysis has resulted.

Coudray has thus interpreted his results: paraldehyde first affects the grey substance of the cerebral hemispheres (sleep, abolition of voluntary movements). It acts on the excito-motor power of the medulla only in toxic doses, and then reflex movements disappear with the voluntary, and the contraction of the pupil proves that the nervous system is deeply affected. General anesthesia is also observed.

The above action of paraldehyde explains its antagonism to strychnine; and experience has proved that not alone are the two substances antagonistic, but that paraldehyde administered in time and in sufficient dose, is the antidote of strychnine even in lethal amount.

The secondary effects of paraldehyde are diminution of arterial tension, slowing of the beats of the heart and of the respiration (and this is only seen after very large doses), increase of the salivary secretion, and almost total elimination by the respiratory tract, the special odor of the drug being apparent.

Therapeusis.—Cervello first used paraldehyde in the in-

somnia of the insane, and of ninety cases he failed in only seven, these being instances of melancholia or of general paralysis. 30 grains secured sleep of five to seven hours' duration. All alienists now recommend it in cases where agitation and insomnia prevail and where it is requisite to obtain rapid effects.

In healthy individuals Desnos, who has carefully reported thirty-five observations, has only exceptionally seen digestive disturbances. He says that paraldehyde is a powerful hypnotic, less anesthetic than chloral, but not likely to affect the heart as the latter is, unless given in large dose, and therefore it may be given to cardiac and debilitated subjects.

According to the same writer paraldehyde has no influence on fever, and the healthy sleep which it procures has no bad effect on the course of the affection.

Notwithstanding the success in some cases of neuralgia we must admit that the drug is inferior to chloral where pain is present.

The relaxing properties of paraldehyde have been utilized in case of tetanus, where it succeeded after chloral had failed; it might be used to advantage in strychnine poisoning.

Keraval and Nercam have stated that paraldehyde was of use in the convulsive neuroses, and a personal observation would seem to prove this. In an epileptic of fifty-two the drug was given daily for fifteen days in the dose of 37½ grains, and the attacks were suspended for sixy-three days, whereas before they had recurred regularly once each week; I have already stated under acetanilid that I succeeded similarly in the same patient.

Finally, to resume the action of paraldehyde, it is a precious hypnotic on account of its rapidity and certainty, but tolerance is soon established.

Further research is necessary to determine its value in relaxing spasm.

Method of Use.—Paraldehyde may be given by the stomach, the rectum, or subcutaneously; the first is preferable.

Abroad and in Italy, doses of ʒ j, ʒ iss and ʒ ij a day are given. In France ʒ j is deemed always sufficient for hypnotic effect; Dujardin-Beaumetz gives 30 grains in order to suppress the initial period of excitability which follows on larger doses. Desnos claims that if hypnotic effects are not obtained from 45 to 60 grains, it is useless to push it.

When paraldehyde is given for analgesic effects in neuralgia, the larger doses may be administered.

The disagreeable taste of paraldehyde may be disguised by the following formula:

Paraldehyde, ...	gr. xxx.
Syr. menth. pip., ...	ʒ vj gr. xv.
Muc. acaceæ, ...	℥ iij ʒ iiss.

By the rectum it may be given as follows:

Paraldehyde, ...	ʒ j to ʒ j gr. xv.
Yolk of egg, ...	No. 1.
Water, ...	℥ iv.

Hypodermically the following formula is useful:

Paraldehyde,		
Aq. lauro-ceraseæ,		
Aq. distillat.,	āā ʒ j gr. xv.	ʒ ij gr. xxx.

The great objection to the hypodermic use is the number necessary for effect.

Pelletierin.

The bark of the root of the pomegranate has for long enjoyed the reputation of being a tenifuge, but its active principle was only isolated in 1878 by Tanret, who gave it the name of pelletierin.

Pelletierin ($C^{16}H^{15}NO^{2}$) is a white liquid, changing color rapidly on exposure to the air, very little soluble in water, very soluble in alcohol and ether, of the density of 0.988.

Physiological Effects.—Numerous experiments on animals, and in particular those of Dujardin-Beaumetz, prove that the alkaloids of the pomegranate belong to the same class of poisons as woorara. Without affecting sensibility, they paralyze the functions of the motor nerves, leaving muscular contractility intact; this action is localized in the terminal fibres of the musculo-motor nerves, and the same effects may be obtained as from woorara.

Therapeusis.—There exists a notable difference in the action of the sulphate and the tannate of pelletierin given in therapeutic doses. Both give rise to a little vertigo and to

transient ocular disturbances, to a hyperemia of the fundus oculi connected with a general encephalic congestion. Further, there occur nausea and at times vomiting; but these phenomena are lacking with the tannate. The tannate being less soluble, remains longer in the intestine, whence more prolonged action. The sulphate has the disadvantage of determining a more pronounced paresis of the intestine, and therefore it opposes the evacuations which are necessary for the complete expulsion of the teniæ.

It is to-day a well-established fact that the administration of a salt of pelletierin causes the expulsion of the entire tenia. The following is considered to be the method of action of this agent: It prevents the tenia from attaching itself for a while to the intestine; this action, however, is transient, and it is, therefore, essential to follow the pelletierin with a cathartic which will provoke contraction and expel the parasite; if we are slow in giving this purgative the result is imperfect, the head remaining behind. Then it is well not to attempt dislodgement again for two to three months.

The tenifuge property of pelletierin is the only one which has as yet been utilized.

Method of Administration.—The dose of the tannate is 22½ grains; it should be given as follows: at night a large enema should be ordered, milk being the sole diet for supper; in the morning the dose of pelletierin is taken fasting, dissolved in fifty times its weight of water acidulated by tartaric acid. About one hour afterwards, an energetic drastic purgative should be given.

Rarely does this drug fail.

Resorcin.

Resorcin ($C^{12}H^{6}O^{4}$) is a composite organism, shining white, crystallizing in long, fine needles, slightly like phenic acid in odor, sweet in taste, a trifle aromatic. It is very soluble in water, in alcohol, and in vaselin.

Antiseptic Properties.—A 1 per cent. solution of resorcin stops or prevents fermentation by destroying the ferments. This action has been proved by Dujardin-Beaumetz, who, with Callias, demonstrated the anti-putrid properties of this substance. Taking pieces of the spleen or of the pancreas of sub-

jects dead of typhoid fever, these writers were able to preserve them indefinitely in a 1.50 per cent. solution of resorcin. These same pieces having been plunged in water when already putrid, the addition of resorcin in 1.50 per cent. caused in a few days the disappearance of odor as well as of the microbes which had been present. Added to urine in the proportion of 1 per cent. resorcin completely checks the ammoniacal fermentation.

These anti-fermentative properties of resorcin class it with the phenols.

Physiological Properties.—Strong solutions of resorcin are local irritants, but below one to twenty the irritation is insignificant.

Absorption of the drug in slight amount is well borne, but when in excess of 4½ grains to 2¼ pounds of weight of animal, Dujardin-Beaumetz and Callias observed epileptiform tremblings persisting for a couple of hours and more marked in the limbs than in the trunk. A lethal dose causes rapid convulsions; the respiration, at first anxious and accelerated, diminishes in frequency and stops before the cardiac contractions, which remain accelerated to the end.

Our knowledge of the effects of resorcin on healthy man is due to Andrer and to Péradon, who experimented on themselves.

In small dose the drug causes disturbances of the nervo-muscular system, and if the dose be increased the movements become uncertain, and vertigo, hallucinations and delirium set in. Very large doses (ʒ ij to ʒ iiss) cause disturbances of vision, of the ear, and sleep, which disappear quickly after a rapid appearance; the circulatory and respiratory functions are but little modified, and the digestive tract tolerates the drug well.

Abundant perspiration corresponding to a fall of temperature follow quickly on a dose of 15 to 45 grains, but the substance is chiefly eliminated by the kidneys, and this occurs quickly, which accounts for the absence of cumulative effects and the transitory nature of the physiological phenomena.

Therapeusis.—Resorcin has been used externally and internally.

Externally it is called for on account of its antifermentative and antiseptic properties. Atonic wounds are favorably influenced by resorcin, which stimulates them to cicatrization.

Leblond and Fissueux, experimenting with resorcin and iodoform in the treatment of soft chancre in the female, preferred the former. Andrer cured an anthrax of the arm by covering the pustules with an ointment of equal parts of resorcin and of vaselin.

Dujardin-Beaumetz cured an ulcer of the tonsil which had proved intractable to chlorate of potass by touching it with a concentrated solution of resorcin in glycerin. The same gentleman has used resorcin with benefit as an antiseptic in vaginal and uterine affections.

As a urethral injection resorcin has proved effective in gonorrhœa, to the same extent as the permanganate of potass, the sulphate of quinine, or sublimate.

In general surgery resorcin takes the place of phénic acid, being inodorous and at least as efficacious. From the latter stand-point we believe that the drug does not as yet occupy the place it should in surgery.

The internal administration of resorcin has been recommended on account of its antithermic properties, and it has been thus used in typhoid fever, in articular rheumatism, in hectic fever, in attacks of malaria. But other more powerful antipyretics are preferable, especially since very large doses of resorcin would have to be given in order to obtain an effect which at best is doubtful.

The anti-putrid properties have been with much greater show of reason utilized—

In catarrhal affections of the urinary passages, where change in the urine is to be feared from long sojourn.

In gastric catarrh, and dilatation of the stomach, lavage of the organ by 1 to 200 solutions has given good results to Andrer and others.

In intestinal catarrh resorcin modifies and disinfects the intestinal secretions.

Leblond, Besnier and others have happily used resorcin in angina diphtheritica, either by penciling with solutions of 1 to 15 in glycerine or by pulverizations with an aqueous solution of 1 to 100.

Finally Moncorvo lauds applications to the larynx of a 1 to 100 solution in case of whooping-cough; according to him we thus rapidly diminish the number and intensity of the attacks and abridge the duration of the disease.

Method of Administration.—The best internal formula for the administration of resorcin is:

Resorcin, ...	gr. xv to ʒ j gr. xv.
Aq. lauro-cer., . . .	ʒ j gr. xv.
Syr. aurant. flor., . . .	℥ j.
Aquæ	℥ iij.

Externally we may use a solution of 1 to 2 per cent. for sprays.

A solution or an ointment of 5, 10 or 20 per cent. for dressings.

A mixture of 1 to 15 in glycerin for wounds and mucous membranes.

For urethral injections 1 to 2 per cent. solutions are strong enough.

SALOL.

In 1883, Nencki, of Berne, discovered salol, and a short time afterwards Sahli introduced it into therapeutics.

Salol, derived from salicylic acid by the substitution of one radicle of phenol for one atom of hydrogen, is a white powder, greasy to the touch, of an agreeable aromatic odor, insipid, insoluble in water, but soluble in alchol.

Soon after the appearance of the essays at Berne, Lépine, of Lyon, used the new drug with his pupil Dr. Montange, and lately Dr. Lombard, a pupil at the Cochin hospital, has published a very interesting thesis.

Physiological Action.—On the temperature salol acts as follows: It constantly lowers the normal figure in the healthy animal; this lowering of temperature occurs after a constant fashion; the fall, after a few limited oscillations, is very rapid, and occurs within a few minutes, then after a slight progressive descent the mercury ascends slowly; the antipyretic effect is not proportionate to the dose. When a certain fall of temperature has been secured, a further dose will not cause a fall proportionate to the amount ingested; even after two consecutive doses lowering of temperature will no longer be obtained by the administration of salol.

It would thus seem as though the organism, after a certain dose of salol, was incapable of reacting further, because a por-

tion alone produces a certain effect, the remainder not being absorbed or resting inert. Experience has proved the truth of this hypothesis, for after the administration of large doses of salol, this drug has been found unaltered in the stools of the animals experimented upon. It further results that salol is not toxic, since the amount absorbed cannot exceed certain limits.

On the respiration the influence of salol is very rapid; a few minutes after the ingestion of this drug, the frequency of the respiratory rhythm increases notably while the amplitude diminishes. But this action is transient, and at the end of an hour the natural order is restored.

As for the circulation, it is scarcely or not at all influenced by salol; the same holds true for the composition of the blood.

How now does salol act? Dr. Sahli first expressed the opinion that salol only decomposed in the presence of the pancreatic juice, and that therefore the amount absorbed would depend on the amount of pancreatic juice secreted. Lépine has made a number of experiments which confirm this view. The chief point of these experiments is the following: If in an animal the course of the pancreatic secretion is turned away from the intestine, the ingested salol passes unchanged into the stools and the reactions of salicylic and of phenic acids are not found in the urine. Salol then reaches the duodenum unaltered, and it is there, in the presence of the pancreatic juice, that it breaks up into salicylic and phenic acids. The quantity absorbed it is difficult to state, but to judge from the effects, it is probably small and subordinate to the proportion of pancreatic juice secreted, this latter varying according to the physiological and the pathological conditions of the individual. This explanation accounts perfectly for the fact that the effects of salol are not in proportion to the dose ingested, and that it is not toxic except under most extraordinary conditions.

The presence of bile, by favoring solution of the salol assists its breaking up by the pancreatic juice.

The products of salol are eliminated by the urine, and these are found in the urine as salicyluric and phenyl-sulphuric acids.

Therapeusis.—From the many experiments made at the Lyon clinic, at Lourcine and at Cochin, it results with certainty that salol quiets pain, and from this stand-point it has

been used with too much enthusiasm. About two hours after the injection of salol, the pains of rheumatism are quieted, but this sedation is of short duration; it ceases usually at the end of twelve to fifteen hours, a trifle longer in case of sub-acute rheumatism. This is the weak point of salol compared with the salicylate of soda, the effect of which is more durable, and this latter possesses the further advantage of attacking the disease as well as the pain.

It is well to reserve salol for the mild uncomplicated cases; the agent may become very valuable for the treatment of rheumatism in children, the great nervous impressionability of whom renders difficult if not dangerous the use of salicylate of soda.

Dr. Lombard thus resumes his conclusions in regard to the use of salol in rheumatism:

As a specific in rheumatism salol is inferior to salicylate of soda. It has no appreciable influence on the progress of the disease.

Salol quiets the pain rapidly, but its action here is more superficial, less durable than that of salicylate of soda.

On account of its evident effect on the pain, and on account of its absolute harmlessness, salol has been and may be used usefully in sub-acute rheumatism, and its use appears legitimate in certain cases of infantile rheumatism.

In typhoid fever salol has been used with about negative effects, and it has no effect on the temperature curve or the progress of the disease; it lowers momentarily the temperature to a few tenths of a degree, but this action is transient and often unperceived. We must add that the same holds true of all the salicylic series.

In certain diseases of the central nervous system, and notably in sclerosis, salol has appeared to diminish appreciably the pain.

In surgery the antiseptic properties of salol have been utilized, and Veuillet has recommended tampons of salolized cotton for use in the cervix. Salol has also proved useful in fungosities of the cervix and in vaginitis.

In external affections of the eye salol seems to be a good adjuvant to boracic acid.

Method of Administration.—Internally salol may be given in cachets in the dose of ʒ j to ʒ iiss for an adult, or in

an emulsion. It may also be mixed with sugar flavored with vanilla.

Externally it may be used mixed with equal parts of starch powder or incorporated in gauze.

A salol collodion has also been prepared as follows:

Salol,	
Ether, ...	āā ʒ j.
Collodion elastic, ...	℥ j.

This is useful in chapped nipples.

SPARTEIN.

Spartein is a volatile alkaloid from genista scoparia of the family of the Leguminosæ, genus Papilionaceæ. It is a colorless liquid of very bitter taste and of a peculiar odor, recalling that of pyridin. It is heavier than water, and its boiling point is rather high; exposure to the air turns it brown. It is little soluble in water, quite so in alcohol, ether and chloroform.

In combination with acids spartein crystallizes with difficulty; the only salt of value therapeutically is the sulphate.

Therapeusis.—First studied by Laborde, who recognized its action on the heart, Professor Sée has noted its utility in cardiac disease; abroad Voigt, Maslowski, Prior and others have also tested it, and the following deductions may be stated in regard to it.

In a healthy man spartein usually increases the amount of urine without affecting the general circulation; still, it has not been proved that this diuretic action is due to a local influence exercised on the epithelium of the kidney, and all goes to prove that it results from direct action on the heart and of modification in intra-vascular tension.

Spartein has produced happy effects in very different cardiac diseases, but with marked differences; thus it is much less efficacious in affections of the muscular substance of the heart than in lesions of the orifices complicated by lack of equilibrium in the compensation.

When, as the result of circulatory disturbances, the renal secretion is lessened, spartein effectively re-establishes diuresis and regularizes the contractions of the heart, and thence there is rapid disappearance of edema and dropsy.

The intra-vascular tension being increased, the frequency of the pulse diminishes as the cardiac rhythm becomes more regular; and even in cases where spartein seems unable to regulate the heart action, it has been observed that it exercises a favorable influence on the dyspnea and distress.

Spartein is active about two to three hours after its administration, and its influence varies from a few hours to two to three days; it is not cumulative.

Spartein, then, should be employed in:

Those affections of the heart where digitalis has proved inefficacious and seems to be contra-indicated. Cases where it is urgent to regulate as promptly as possible and to increase the action of the heart. Diseases which are accompanied by diminution of the urinary secretion.

Method of Administration.—At the outset the dose should vary from ⅓ of a grain to 1½ grains daily; but G. Sée has shown that this is too little, and advises the administration at once of 1½ grains, and the repetition of this dose two to three times daily.

The following formula is commended:

Aquæ, ...	℥ j ʒ iij.
Aq. lauro-ceris, ...	ʒ iij.
Syr. simplicis, ...	ʒ v.
Spartein sulph., ...	gr. viiss.

It may also be given in pills containing 1½ grains.

Strophanthus Hispidus.

Strophanthus hispidus belongs to the family Apocyneæ, and grows especially in Africa and in Central Asia. Many savage tribes use the juice of the plant to poison the points of their arrows. The seeds, in particular, contain the toxic element of the plant; they are pale yellow or brown in color, of the length of .19 of an inch and .14 to .2 of an inch in thickness.

Physiological Effects.—Pelikan and Vulpian first studied strophanthus, finding in it properties analogous to those of digitalis, black hellebore, etc. (depression of muscular irritability and paralysis of the heart). According to these writers the heart is first affected, and this action once attained the organ never answers to any excitant, mechanical or chemical,

acting on the pneumogastric or sympathetic nerves. The ventricles are first paralyzed, the auricles being only later affected.

Strophanthus is far more poisonous than digitalis, and the toxic effects are produced by absorption through the circulation.

As for the action of strophanthus on the muscular system they are altogether comparable to those which follow on the absorption of veratrin; at first there is a stiffening of the muscles, slowness in contraction, and then tetanus and death. At autopsy the veins and the auricles are found gorged with blood, while the ventricles and the arterial system are absolutely empty.

The effects of the agent given in toxic doses are very rapid, and in mammiferæ they occur in two to five minutes after the injection of $\frac{1}{6}$ of a grain of the alcoholic extract; the signs of poisoning are the following: malaise, dyspnea with irregularity of the respiratory rhythm, weakness, vomiting with intestinal cramps, coma, convulsions, sudden death.

Sometimes the following additional symptoms occur: muscular tremor, syncope, pain like that of angina.

The toxic dose of strophanthus is not at all proportionate to the size of the animal, and even in mammiferæ astonishing idiosyncrasies are met with. The toxic property of the agent is communicated to the blood of the animal experimented upon, and Polaillon and Carvilles have been able to poison animals by the injection of a syringe-full of the blood of one of the poisoned animals.

The different observers admit that strophanthus acts but little, if at all, on the nerves, and that the intelligence is preserved in the midst of the general disturbances of poisoning.

Strophanthus, then, is an exclusively muscle poison, acting on both the striated and the non-striated fibres, and this is why it is especially a heart poison, since this organ is the muscle which receives proportionately the most blood—this being the vehicle of the poison.

Therapeusis.—It was in the disturbances of compensation consecutive to a valvular affection that Fraser first tested the action of strophanthus, and in cases where the myocardium is not diseased the action is very remarkable; at the end of a few minutes the sphygmographic tracing is modified, and the

energy of the cardiac contractions is increased; the pulse becomes strong and regular, and in cases of complete asystole with pulmonary edema and generalized anasarca Fraser has obtained the disappearance of these symptoms in about one week; the tracings published by this author are very interesting and conclusive.

Coincidently with the disappearance of the dropsy there is a considerable increase in the urine; this latter action is due purely to the influence of the agent on the central organ of the circulation, for the experiments of Fraser, of Polaillon and Carville prove that strophanthus has scarcely any action on arterial contractility, and this constitutes a marked difference between the effects of this agent and those of digitalis.

Strophantin given hypodermically causes still more rapid effects.

The patients feel better at once, even before the objective signs have been altered for the better. Fraser has observed that the effects of strophantin are lasting, and, therefore, the result aimed at once gained the doses may be given at infrequent intervals.

Poulée has used strophanthus in the nephritis following on scarlet fever; he speaks very favorably of it here, and reports a number of conclusive cases; the same writer recommends strophanthus in hemorrhage accompanying vaso-constrictor phenomena, and whenever it is desired to lower the tension and the rhythm of the circulation, as in the metrorrhagia of the menopause.

According to Puis, of Vienna, strophanthus is also indicated in pyrexia leading to paresis of the myocardium, and Poulet administers it as a powerful antiphlogistic.

In local paralysis, of peripheral origin, Poulet has obtained excellent results, and he states the following conclusion:

"Strophanthus, owing to its effect on the muscular system, is of great value in paralysis. Not being excitant, like strychnine, it may be used to advantage in the first stage of the disease where nux vomica cannot."

Method of Administration.—The active principle of strophanthus is chiefly contained in the seeds, and Fraser gave from three to four drops daily of the tincture prepared from these. In France a diluted tincture is prepared, of which eight to twelve drops may be given.

Two alkaloids have been obtained from strophanthus: inein, almost inactive and not used, and strophantin, very toxic, and which should always be first given in the dose of $\frac{1}{250}$ of a grain; it is with this alkaloid that Fraser has always obtained his best results. Poulet gives the powder of the seeds in pill form in the dose of $1\frac{1}{2}$ to 3 grains.

The stomach is the best route of administration, and only in very urgent cases should the agent be given hypodermically.

Terpin and Terpinol.

Terpin ($C^{10}H^{16}+6H^{2}O$) is a derivative of turpentine, having about the same therapeutic properties, only to a higher degree. Introduced into materia medica in 1886 by Lépine, it has been studied by G. Sée, and since numerous observers have used and praised it in the treatment of pulmonary, bronchial and renal affections.

As for terpinol, which is obtained by the action of acids on terpin, Dujardin-Beaumetz claims that it has a powerful action on the respiratory tract, whence it is eliminated.

Physiological Effects.—In a high dose terpin provokes nausea and a little diarrhea. In the dog Lépine has found a diminution of the urinary secretion, albuminuria and even hematuria; the respiration is accelerated and irregular, the temperature rises, and in toxic dose the phenomena are the same as those of turpentine.

Therapeutic Effects.—Lépine has used terpin in neuralgia, and he was successful with this substance even in a case of hystero-epilepsy.

As a modificator of the renal functions there is less satisfaction with terpin than Lépine had at the start, and turpentine seems to be more useful; in chronic nephritis, if more than $7\frac{1}{2}$ grains are given, we are apt to provoke hematuria.

The true usefulness of terpin, and possibly the sole one which entitles it to a rank in the materia medica, is as a modificator of the bronchial secretions. In small dose terpin increases and makes fluid the bronchial secretions, while in large dose it diminishes them considerably or even checks them. Lépine experimented almost entirely with the first of these properties.

Germain Sée, on the contrary, recommends a large dose, and his conclusions are the following:

"Terpin is an energetic modifier of the respiratory mucous membrane and a powerful anti-secreting agent; it diminishes and rapidly checks expectoration in the catarrhal forms of phthisis, whether the secretion comes from the bronchi irritated by the tubercles or from the walls of the pulmonary cavities; whether the disease is in its first stage or in the purulent, or in the stage of excavation, terpin is indicated whenever the formation of pus is abundant enough to fatigue the patient or to wear him out; it may be employed with success in the hemoptysis of beginning tuberculosis—that is to say, when the disease has not reached the stage of cavity formation with aneurisms of the pulmonary artery; in the treatment of chronic bronchitis independent of asthma it is the best means of lessening bronchial hyper-secretion, and its certain action should cause it to be preferred to turpentine, etc., and on account of its harmlessness it has advantages over creasote."

In nervous, emphysematous or catarrhal asthma terpin must yield to iodine and to pyridin.

To resume: Terpin, although it should be reserved for a restricted class of diseases, is still a valuable agent, with an honorable place in the materia medica.

Method of Administration.—Terpin being only slightly soluble in water, an alcoholic solution may be used when alcohol is not contra-indicated; otherwise about eight ounces of non-alcoholic liquid is needed for the solution of 15 grains of terpin; if the agent causes diarrhea, an astringent syrup should be added to it.

In bronchorrhea I have used the following pills with special effect:

Terpin, ...	gr. iss.
Creasote,	
Eucalyptol, ...	āā gr. ¾

Three to six pills are to be taken daily.

When we wish to increase the fluidity of the secretions and to facilitate their expectoration, doses of 6 to 7½ grains will suffice; but if we wish to check the muco-purulent secretion 15 grains must be given, and even 30 grains may be given without fear.

Terpin had best be ordered at meal-time, in order to avoid possible digestive troubles.

Terpinol is less used than terpin, and is given in capsules containing 1½ grains. From 10 to 20 of these may be ordered daily.

THALLIN.

Thallin was recently discovered by Skraus, of Vienna, and introduced into therapeutics by Jaksch; its chemical name is tetra-hydroparaquinizol, and its formula is C^9H^6, NH^4, CO, CH^3.

The name thallin is due to the fact that it turns emerald green when brought in contact with the per salts of iron. It is an oily liquid, and may be transformed into a crystalline substance with an odor like that of coumarin.

The sulphate is the only salt used. It is a crystalline powder, of disagreeable, bitter, saline taste, of the odor of anisol, little soluble in water and less in alcohol.

Therapeusis.—As an antiseptic it has not given much result; its most certain action is to lower the temperature. This occurs in from two to four hours; antipyresis is considerable but transient, and in order to keep up the effect it is necessary to repeat the dose about every two hours. When the agent is stopped the temperature rises, with chills and perspiration.

Thallin is rapidly absorbed, but its elimination is slow. The advantages it has over antipyrin is the absence of digestive disturbance, and the small doses in which it need be given for antipyretic effect: but then it is less certain in action, it is necessary to repeat the dose frequently, and it lowers intravascular pressure; these objections have stood in the way of its routine use.

Method of Administration.—Thallin may be given in solution or in pills: the daily dose varies from 3 to 15 grains. The best method is to give 3 grains and then 1½ grains every three hours. A shorter interval might cause dangerous thermometric reaction.

URETHAN.

Urethan or ethylurethan is the ethylic ether of carbonic acid, studied by Würtz and Rose. This agent, which possesses hypnotic properties, was introduced into therapeutics by Schmiedeberg, and principally studied in France by Hu-

chard. It appears as white crystals, with scarcely any odor and of a special taste; it is very soluble in water, alcohol and ether.

Physiological Action.—Jacksch has used this agent in a large number of cases, and has determined that it causes sleep without noteworthy disturbance of the respiration or of the circulation. In small dose it acts but slowly, and after repeated doses only, while in doses of 15 grains it acts almost instantaneously. Jacksch claims that it acts on the nervous centres to the exclusion of the peripheral nerves, and this would explain its lack of efficacy in neuralgias of the latter class.

Huchard states that urethan causes a calm sleep without consecutive digestive or cephalic disturbances. Sleep ensues in from ten minutes to one hour after administration, and lasts for from four to six hours. The vascular tension is not modified.

Urethan is only slightly toxic.

Therapeusis.—Chauson, who has specially studied this substance, claims that urethan is indicated whenever we desire to act on the cord to diminish its reflex excitability, and he says he has succeeded several times in nocturnal incontinence of urine.

In tetanus, urethan has the advantage over chloral that it may be continued a long time and in high dose without causing disturbance.

In chorea, also, this agent has seemed of utility.

As a hypnotic urethan seems indicated whenever insomnia is due to some medullary trouble, or when we wish to avoid the secondary effects of other hypnotic agents, such as chloral. It is very useful in cardiacs, and seems to have a certain advantageous influence on the diseased organ.

Whenever, however, the cause of the insomnia is not reflex from the cord, chloral is preferable to urethan.

In phthisis urethan is superior to opiates; it diminishes the dyspnea and the cough. It is equally of use in the insomnia of alcoholism, of dyspeptics, of the debilitated.

Method of Administration.—In the adult 30 to 60 grains must be given at once. In the infant 1½ grains suffice at one year; afterwards the dose may be increased in proportion to the age, as is customary with other active agents.

LIQUID VASELIN. (*Medicinal.*)

This is a mineral oil obtained by distillation from *Oléophates russes.* The name vaselin is not appropriate, since it contains none; it is used in the arts for greasing machinery; it is purified for medicinal purposes.

It has been called naphtolein, oleonaphtin, etc.; the name liquid vaselin has been given it in France. Balzer introduced it into therapeutics, having procured it in the pure state and having used it as a solvent for salts of mercury.

Properties.—Medicinal liquid vaselin is a colorless, odorless mineral oil, insipid, perfectly neutral in the pure state; it is not oxidizable, and therefore it may be preserved indefinitely. It dissolves the essential oils in equal parts, and in variable proportions a number of other bodies: iodine, sulphur, iodoform, phenol, etc. By means of a special process it can be made to dissolve a number of alkaloids.

Liquid vaselin may be injected hypodermically with impunity, and its ready diffusibility in the organism causes the agents incorporated with it, whether in solution or in suspension, to pass into the system, so that in certain instances, the patient tastes it and traces are found in the urine.

Vaselin thus introduced into the system has been found innocuous; owing to the fact that it is not oxidizable, it is preferable to other essential oils, although phenic acid is soluble in greater proportions in the latter.

Without entering into details we content ourselves with enumerating the most important formulæ given by A. Meunier:

1.	Eucalyptol pur.	5 parts.
	Vaselin pur.	20 "
	Dose: gr. xv. to ʒ iij gr. xlv daily.	
2.	Eucalyptol, ...	5 parts.
	Iodoform, ...	25 "
	Liquid vaselin, ...	20 "
	Same dose as preceding.	
3.	Sulphide of carbon, ...	1 part.
	Liquid vaselin, ...	19 parts.
	Dose: gr. xv. to xxx. in fractions.	

4.	Terebinth. pur.	5 parts.
	Liquid vaselin	20 "
	Dose: gr. xv. to ʒ iiss daily.	
5.	Menthol	10 parts.
	Vaselin	90 "
6.	Thymol.	1 part.
	Vaselin	200 parts.
7.	Phenol	1 part.
	Vaselin	100 parts.
8.	Iodine	1 part.
	Vaselin	100 parts.
9.	Camphor	1 part.
	Vaselin	100 parts.
10.	Eugenol	3 parts.
	Vaselin.	100 "
	(Eugenol is considered a powerful microbicide.)	
11.	Helenin	1 part.
	Vaselin	100 parts.
12.	Chloroform	20 "
	Vaselin	80 "

Alkaloids are first dissolved in chloroform and then mixed with vaselin; the dose varies according to the toxicity of the agent and the effect which is desired.

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